

## SECTION 4

### TARGET ANALYTES

The selection of appropriate target analytes in fish and shellfish contaminant monitoring programs is essential to the adequate protection of the health of fish and shellfish consumers. The procedures used for selecting target analytes for screening studies and a list of recommended target analytes are presented in this section.

#### 4.1 RECOMMENDED TARGET ANALYTES

Recommended target analytes for screening studies in fish and shellfish contaminant monitoring programs are listed in Table 4-1. This list was developed by the EPA 1993 Fish Contaminant Workgroup from a review of the following information:

**1. Pollutants analyzed in several national or regional fish contaminant monitoring programs—**The monitoring programs reviewed included

- National Study of Chemical Residues in Fish (U.S. EPA)
- National Dioxin Study (U.S. EPA)
- 301(h) Monitoring Program (U.S. EPA)
- National Pollutant Discharge Elimination System (U.S. EPA)
- National Pesticide Monitoring Program (U.S. FWS)
- National Contaminant Biomonitoring Program (U.S. FWS)
- National Status and Trends Program (NOAA)
- Great Lakes Sportfish Consumption Advisory Program
- National Water Quality Assessment Program (USGS).

Criteria for selection of the target analytes in these programs varied widely depending on specific program objectives. The target analytes used in these major fish contaminant monitoring programs are compared in Appendix E. Over 200 potential contaminants are listed, including metals, pesticides, base/neutral organic compounds, dioxins, dibenzofurans, acidic organic compounds, and volatile organic compounds.

Table 4-1. Recommended Target Analytes

Metals	Organophosphate Pesticides
Arsenic (inorganic)	Chlorpyrifos
Cadmium	Diazinon
Mercury (methylmercury)	Disulfoton
Selenium	Ethion
Tributyltin	Terbufos
Organochlorine Pesticides	Chlorophenoxy Herbicides
Chlordane, total (cis- and trans-chlordane, cis- and trans-nonachlor, oxychlordane)	Oxyfluorfen
DDT, total (2,4'-DDD, 4,4'-DDD, 2,4'-DDE, 4,4'-DDE, 2,4'-DDT, 4,4'-DDT)	PAHs <sup>d</sup>
Dicofol	PCBs
Dieldrin	Total PCBs <sup>e</sup> (sum of PCB congeners or Aroclor equivalents)
Endosulfan (I and II)	Dioxins/furans <sup>f,g</sup>
Endrin	
Heptachlor epoxide <sup>a</sup>	
Hexachlorobenzene	
Lindane ( $\gamma$ -hexachlorocyclohexane; $\gamma$ -HCH) <sup>b</sup>	
Mirex <sup>c</sup>	
Toxaphene	

PAHs = Polycyclic aromatic hydrocarbons; PCBs = Polychlorinated biphenyls; DDT = p,p'-dichlorodiphenyl trichloroethane; DDE = p,p'-dichlorodiphenyl dichloroethylene; and DDD = dichlorodiphenyldichloro ethane.

<sup>a</sup> Heptachlor epoxide is not a pesticide but is a metabolite of two pesticides, heptachlor and chlordane.

<sup>b</sup> Also known as  $\gamma$ -benzene hexachloride ( $\gamma$ -BHC).

<sup>c</sup> Mirex should be regarded primarily as a regional target analyte in the Southeast and Great Lakes states, unless historic tissue, sediment, or discharge data indicate the likelihood of its presence in other areas.

<sup>d</sup> It is recommended that tissue samples be analyzed for benzo[*a*]pyrene, and 14 other PAHs and that the order-of-magnitude relative potencies given for these PAHs be used to calculate a potency equivalency concentration (PEC) for each sample for comparison with the recommended SVs for benzo[*a*]pyrene (see Section 5.3.2.5).

<sup>e</sup> Analysis of total PCBs (as the sum of Aroclors or PCB congeners) is recommended for conducting human health risk assessments for total PCBs (see Sections 4.3.6 and 5.3.2.6). A standard method for Aroclor analysis is available (EPA Method 608). A standard method for congener analysis (EPA Method 1668) is currently under development; however, it has not been finalized. States that currently do congener-specific PCB analysis should continue to do so and other states are encouraged to develop the capability to conduct PCB congener analysis. When standard methods for congener analysis are verified and peer reviewed, the Office of Water will evaluate the use of these methods.

<sup>f</sup> Note: The EPA Office of Research and Development is currently reassessing the human health effects of dioxins/furans.

<sup>g</sup> It is recommended that the 17 2,3,7,8-substituted tetra- through octa-chlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs) and 12 dioxin-like PCBs be determined and a toxicity-weighted total concentration calculated for each sample (Van den Berg et al., 1998) (see Sections 4.3.7, 5.3.2.6, and 5.3.2.7).

**2. Pesticides with active registrations**—The EPA Office of Pesticide Programs (OPP) Fate One Liners Database (U.S. EPA, 1993a) containing information for more than 900 registered pesticides was reviewed to identify pesticides and herbicides with active registrations that met four criteria. The screening criteria used were

- Oral toxicity, Class I or II
- Bioconcentration factor greater than 300
- Half-life value of 30 days or more
- Initial use application profile.

At the time of this review, complete environmental fate information was available for only about half of the registered pesticides. As more data become available, additional pesticides will be evaluated for possible inclusion on the target analyte list.

Use of the OPP database was necessary because many pesticides and herbicides with active registrations have not been monitored extensively either in national or state fish contaminant monitoring programs.

3. **Contaminants that have triggered states to issue fish and shellfish consumption advisories or bans**—The database, *National Listing of State Fish and Shellfish Consumption Advisories and Bans* (RTI, 1993), was reviewed to identify specific chemical contaminants that have triggered issuance of consumption advisories by the states. As shown in Table 4-2, four contaminants (PCBs, mercury, chlordane, and dioxins/furans) triggered advisories in the largest number of states in 1993. As a comparison, the number of states issuing advisories for each pollutant in 1998 has also been presented while the total number of states issuing advisories for most pollutants generally has increased, the number of states issuing advisories for two major pollutants, chlordane and dioxin, has decreased over the past 5 years.
4. **Published literature on the chemistry and health effects of potential contaminants**—The physical, chemical, and toxicologic factors considered to be of particular importance in developing the recommended target analyte list were
  - Oral toxicity
  - Potential of the analyte to bioaccumulate
  - Prevalence and persistence of the analyte in the environment
  - Biochemical fate of the analyte in fish and shellfish
  - Human health risk of exposure to the analyte via consumption of contaminated fish and shellfish
  - Analytical feasibility.

Final selection of contaminants by the EPA 1993 Workgroup for the recommended target analyte list (Table 4-1) was based on their frequency of inclusion in national monitoring programs, on the number of states issuing consumption advisories for them in 1993 (Table 4-2), and on their origins, chemistry, potential to bioaccumulate, estimated human health risk, and feasibility of analysis. Primary consideration was also given to the recommendations of the Committee on Evaluation of the Safety of Fishery Products, published in *Seafood Safety* (NAS, 1991).

### 4.2 SELECTION AND PRIORITIZATION OF TARGET ANALYTES

The decision to conduct a fish tissue monitoring study is normally the result of the discovery of specific contaminants during water quality or sediment studies and/or

**Table 4-2. Contaminants Resulting in Fish and Shellfish Advisories**

Contaminant	Number of states issuing advisories	
	1993	1998
<b>Metals</b>		
Arsenic (total)	1	3
Cadmium	2	3
Chromium	1	1
Copper	1	1
Lead	4	5
Mercury	29	40
Selenium	5	5
Tributyltin	1	0
Zinc	1	1
Organometallics	1	1
Unidentified metals	3	1
<b>Pesticides</b>		
Chlordane	24	22
DDT and metabolites	9	12
Dieldrin	3	6
Heptachlor epoxide	1	1
Hexachlorobenzene	2	2
Kepone	1	1
Mirex	3	3
Photomirex	1	0
Toxaphene	2	4
Unidentified pesticides	2	2
<b>Polycyclic aromatic hydrocarbons (PAHs)</b>	3	4
<b>Polychlorinated biphenyls (PCBs)</b>	32	36
<b>Dioxins/furans</b>	20	19
<b>Other chlorinated organics</b>		
Dichlorobenzene	1	1
Hexachlorobutadiene	1	1
Pentachlorobenzene	1	0
Pentachlorophenol	1	2
Tetrachlorobenzene	2	0
Tetrachloroethane	1	0
<b>Others</b>		
Creosote	2	2
Gasoline	1	1
Multiple pollutants	2	1
Phthalate esters	1	0
Polybrominated biphenyls (PBBs)	1	1
Unspecified pollutants	3	0

Sources: RTI, 1993; U.S. EPA, 1999c.

the identification of pollutant sources in waters routinely used by recreational or subsistence fishers. EPA recognizes that measuring all 25 target analytes in fish tissues collected at all state monitoring sites is expensive and that cost is an important consideration that states must evaluate in designing and implementing

their fish monitoring programs. Ideally, if resources are available to conduct sampling and analysis of all 25 target analytes, the state should consider this option because it provides the greatest amount of information for fishers in the state on levels of contamination statewide. Also, this approach can better detect the presence of those contaminants that are transported long distances from their points of release (e.g., methylmercury, dioxins/furans, toxaphene), often outside the state's borders, and contaminate relatively pristine areas devoid of any obvious pollutant sources.

If the cost of this approach is prohibitive, however, the state may wish to use a watershed-based approach as a way to reduce sampling and analysis costs (Table 4-3). The selection and prioritization recommendations discussed below are watershed-based and take into consideration land use categories (rural, agricultural, suburban/urban, and industrial) as well as geological characteristics, regional differences, and national pollution trends. Land use patterns (both current and historic) are often the most important factors in deciding what analytes to select for analysis. The watershed-based approach gives the highest priority (XXX) to analysis of contaminants that are widely dispersed nationally and relatively inexpensive to analyze, such as mercury. This approach gives a lower priority (X) to monitoring organochlorine pesticides (e.g., chlordane, DDT, and dieldrin) at rural and suburban sites, but a higher priority (XX) to monitoring these same chemicals in agricultural watersheds where their use has been extensive or in industrial watersheds where they may have been released during manufacturing, formulation, packaging, or disposal. Because of the very high cost of analysis for some contaminants (e.g., PCBs and dioxins/furans and dioxin-like PCBs), this watershed approach also allows money for these analyses to be directed toward analysis primarily in suburban/urban and industrial watersheds where sources either from historic manufacturing or historic and/or current practices (combustion or incineration sources) have been identified or where water and/or sediment data in the watershed have detected these chemicals at elevated concentrations.

States should use all available environmental data and their best scientific judgment when developing their fish monitoring programs. Using the watershed approach gives states the flexibility to tailor their sampling and analysis programs to obtain needed information as cost-effectively as possible by directing limited resources to obtaining information on contaminant levels most likely to be found in fish tissue at a given site. To be most effective, states need to recognize and carefully evaluate all existing data when assessing which target analytes to monitor at a particular site. States should include any of the recommended EPA target analytes and any additional target analytes in their screening programs when site-specific information (e.g., tissue, water, or sediment data; discharge monitoring data from municipal and industrial sources; or pesticide use data) suggests that these contaminants may be present at levels of concern for human health.

**Table 4-3. Selection and Prioritization of Target Analytes by Watershed Type**

Analyte	Rural	Agricultural	Suburban/ Urban	Industrial	Sources/Uses
<b>Metals</b>					
Arsenic	XX <sup>a</sup>	X <sup>a,b</sup>	X <sup>a,b</sup>	XX <sup>b</sup>	Naturally occurring as a sulfide in mineral ores; fossil fuel combustion; mining/smelting; wood preservative; insecticide, herbicide, and algicide; hazardous waste site leachate
Cadmium	XX <sup>a</sup>	X <sup>a,b</sup>	X <sup>a,b</sup>	XX <sup>b</sup>	Smelting/mining; surface mine drainage; uses in paints, alloys, batteries, plastics, pesticides, herbicides; waste disposal operations.
Mercury	XXX <sup>c</sup>	XXX <sup>c</sup>	XXX <sup>c</sup>	XXX <sup>c</sup>	Naturally occurring; atmospheric transport from fossil fuel combustion; mining/smelting; chlorine alkali production; historic use in pulp and paper and paints; Hazardous waste site leachate; statewide freshwater and/or coastal advisories in 15 states
Selenium	XX <sup>a</sup>	X <sup>a</sup>	X <sup>a</sup>	XX <sup>d</sup>	Naturally occurring in west and southwest soils; emissions from fossil fuel combustion; leachate from coal fly ash disposal areas
Tributyltin			X <sup>d</sup>	XX <sup>d</sup>	Shipyards and marinas; uses in antifouling paint, cooling tower disinfectants, wood preservatives, pulp and paper industry, and textile mills.
<b>Organochlorine Pesticides</b>					
Chlordane		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Domestic termite control; pesticide manufacturing/packaging/formulation sites
DDT		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Broad spectrum pesticide use; pesticide manufacturing/packaging/formulation sites
Dicofol <sup>e</sup>		XX <sup>b</sup>		XX <sup>b</sup>	Miticide/pesticide for cotton, apples, and citrus primarily in FL and CA; lesser use in turf, ornamentals, pears, apricots, and cherries; pesticide manufacturing/packaging/formulation sites
Dieldrin		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Broad spectrum pesticide for termites/soil insects and for cotton, corn, and citrus; pesticide manufacturing/packaging/formulation sites
Endosulfan <sup>e</sup>		XX <sup>b</sup>		XX <sup>b</sup>	Noncontact insecticide for seed and soil treatments; pesticide manufacturing/packaging/formulation sites
Endrin		XX <sup>b</sup>		XX <sup>b</sup>	Broad spectrum pesticide; pesticide manufacturing/packaging/formulation sites
Heptachlor epoxide		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Degradation product of heptachlor used as a contact and ingested soil insecticide for termites and household pesticide and chlordane also used as a termiticide; pesticide manufacturing/packaging/formulation sites for heptachlor and chlordane

Table 4-3. (continued)

Analyte	Rural	Agricultural	Suburban/ Urban	Industrial	Sources/Uses
Hexachlorobenzene		XX <sup>b</sup>		XX <sup>b</sup>	Fungicide used as seed protectant, used as chemical intermediate in production of many other organochlorine pesticides; pesticide manufacturing/packaging/formulation sites for a wide variety of organochlorine pesticides
Lindane <sup>e</sup>		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Seed and soil treatments for tobacco; foliage applications for fruit and nut trees and vegetables; wood preservative. pesticide manufacturing/packaging/formulation sites
Mirex		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Used extensively in Southeast and Gulf Coast states against fire ants; used in fire retardants and plastic polymerizer; pesticide manufacturing/packaging/formulation sites
Toxaphene		XX <sup>b</sup>		XX <sup>b</sup>	Insecticide for cotton; piscicide for rough fish; pesticide manufacturing/packaging/formulation sites
<b>Organophosphate Pesticides</b>					
Chlorpyrifos <sup>e</sup>		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Widely used on cotton, peanuts, and sorghum as well as fruits and vegetables; domestic household insecticide with lawn and garden applications. Use applications will change by the end of 2001. All residential use will end as will use on tomatoes. Use on apples and grapes will be greatly reduced (U.S. EPA, 2000b). Used as a termiticide in California; pesticide manufacturing/packaging/formulation sites
Diazinon <sup>e</sup>		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Widely used on a broad variety of fruits and vegetables, field crops, and pastureland; domestic household insecticide used for lawn and garden applications; pesticide manufacturing/packaging/formulation sites
Disulfoton <sup>e</sup>		XX <sup>b</sup>		XX <sup>b</sup>	Widely used as a side dressing, broadcast, and foliar spray and as a seed dressing; pesticide manufacturing/packaging/formulation sites
Ethion <sup>e</sup>		XX <sup>b</sup>	X <sup>b</sup>	XX <sup>b</sup>	Major use on citrus, fruit and nut trees, and vegetables. Domestic outdoor use around homes and lawns; pesticide manufacturing/packaging/formulation sites
Terbufos <sup>e</sup>		XX <sup>b</sup>		XX <sup>b</sup>	Used principally on corn, sugar beets, and grain sorghum; pesticide manufacturing/packaging/formulation sites
<b>Chlorophenoxy Herbicides</b>					
Oxyfluorfen <sup>e</sup>		XX <sup>b</sup>		XX <sup>b</sup>	Widely used to control grass and weeds in corn, cotton, soybeans, fruit and nut trees, and ornamental crops; pesticide manufacturing/packaging/formulation sites

Table 4-3. (continued)

Analyte	Rural	Agricultural	Suburban/ Urban	Industrial	Sources/Uses
<b>Polycyclic Aromatic Hydrocarbons (PAHS)</b>			X <sup>d</sup>	X <sup>d</sup>	Components of crude and refined petroleum and coal products; waste incineration, wood preservatives, creosote, coal tar, coal coking, urban runoff from asphalt, automobile tires and exhaust emissions, and petroleum spills; coal gasification sites, and petroleum refineries.
<b>Polychlorinated Biphenyls (PCBs)</b>			X <sup>d</sup>	X <sup>d</sup>	Produced as Arochlors for use as dielectric fluid in electrical transformers and as hydraulic fluid; leachate from land fills and Superfund sites.
<b>Dioxins and Dibenzofurans</b>			X <sup>d</sup>	X <sup>d</sup>	Industrial sites including bleached kraft paper mills, facilities handling 2,4,5-trichlorophenoxyacetic acid (2,4,5,-T), 2,4,5-trichlorophenol (2,4,5-TCP), silvex, hexachlorobenzene, pentachlorophenol, and PCBs.; Industrial and municipal combustors and incinerators

<sup>a</sup> Tissue residue analysis is recommended if geologic characteristics suggest potential for elevated metal concentrations in water or sediment or if sources are identified in the watershed suggesting the presence of this target analyte at the sampling site.

<sup>b</sup> Tissue residue analysis is recommended if use application of this pesticide has been reported in the watershed either from historic or current use data, if sources like pesticide production/packaging/formulation facilities exist in the watershed, or if the state has water and/or sediment data indicating the presence of this target analyte at the sampling site.

<sup>c</sup> Tissue residue analysis is highly recommended at all sites.

<sup>d</sup> Tissue residue analysis is recommended if sources as described in Sources/Uses column are identified in suburban/urban or industrial watershed or the state has water and/or sediment data indicating the presence of this analyte at the sampling site.

<sup>e</sup> Pesticide with currently active registration

**X** = Analysis for target analyte should be considered if water and or sediment analysis results detect the target analyte or if historic or current use information provide evidence for the potential presence of this target analyte in the watershed.

**XX** = Analysis for target analyte is recommended for this land use type if historic or current use information provides evidence of the potential presence of this target analyte in the watershed.

**XXX** = Analysis for target analyte is highly recommended at all stations in all watershed types.

**Rural.** The major analytes of concern in rural waterbodies (i.e., watersheds with no past or current urban/suburban, industrial, or agricultural uses) are the metals, including arsenic, cadmium, mercury, and selenium. Weathering processes in certain geologic areas can result in elevated levels of arsenic, cadmium, mercury, and selenium in water and sediments. State agencies should also be aware of past land use patterns in what are now considered rural areas of their states. For example, abandoned mining sites may be a source of metal contamination via leaching from mine drainage or slag piles. Large areas east of the Appalachians were agricultural watersheds during the early to mid twentieth century. While some of this agriculture land is now suburban/urban in its use, other areas, particularly in the South, are reverting to forests that might at first glance be classified as rural use. Arsenic compounds were used as pesticides in the early

1900s, and, along with organochlorine pesticides, may still be present in farmland abandoned after the 1940s. States should also be aware that mercury has been identified in fish collected from what would be classified as rural or pristine areas of the Great Lakes basins and waterbodies in the northeastern and southeastern states remote from any obvious point sources of pollution. Mercury contamination in these areas seems to be facilitated through the atmospheric transport of this metal. Because mercury is the target analyte that has triggered issuance of the largest number of advisories in the United States (nearly 68 percent of all advisories nationwide) and because of the relatively low cost of chemical analysis for this analyte, EPA recommends that this metal be monitored at all rural sites, especially those where little or no monitoring data are available.

Depending on site-specific conditions and considerations, states may opt to analyze for mercury as well as a suite of other heavy metals that can be analyzed as a group at relatively low cost. The only target analyte metal that should not be analyzed for routinely in rural areas without other supporting data is tributyltin, which is typically found near boatyards and marinas or near wood preservative production facilities. States may include any of the recommended EPA target analytes and any additional target analytes in their screening programs when site-specific information on a rural watershed suggests that these contaminants may be present at levels of concern for human health.

***Agricultural.*** The major analytes of concern in agricultural waterbodies (i.e., watersheds where past or current land use is dominated by agriculture) are the organochlorine and organophosphate pesticides and the chlorophenoxy herbicide, oxyfluorfen. These analytes fall into two categories, those with inactive registrations (i.e. banned or withdrawn from the market) and those with active registrations (endosulfan, lindane, dicofol, chlorpyrifos, diazinon, terbufos, ethion, disulfoton, and oxyfluorfen). Although use of some of the organochlorine pesticides was terminated more than 20 years ago in the United States (e.g., DDT, dieldrin, endrin, and mirex), these compounds still need to be monitored. Many of the organochlorine pesticides that are now banned were used in large quantities for over a decade and are still present in high concentrations at some sites. On a nationwide basis, chlordane and DDT, for example, are responsible for 3 and 1 percent, respectively, of the advisories currently in effect. For the pesticides with active registrations, use and rate application information maintained by the state's Department of Agriculture should be reviewed to identify watersheds where these pesticides are currently used and are likely to be present in aquatic systems as a result of agricultural runoff or drift. Unlike many of the historically used organochlorine pesticides, the pesticides in current use degrade relatively rapidly in the environment. In addition, federal regulations are in effect that set maximum application rates and minimize use near waterbodies. At the time of this writing, no fish consumption advisories for these analytes have yet been issued; however, state agencies should be aware of special circumstances that could result in accumulation in fish. In addition to accidental spills and misapplication, heavy and repeated rainfall shortly after application may wash these pesticides into streams. Signs of pesticide pollution may include erratic swimming behavior in fish as well as fish kills.

It is also important to note that pesticide uses and labels may change over time. All pesticides with active registrations are currently being reviewed by EPA under provisions of the Food Quality Protection Act of 1996. The state agency responsible for designing the fish contaminant monitoring program should be aware of all historic and current uses of each pesticide within its state, including the watersheds, application rates, and acreage where the pesticide has been or currently is applied to ensure that all potentially contaminated sites are included in the sampling plan. Because mercury contamination seems to be facilitated through atmospheric transport, because it has triggered issuance of the largest number of U.S. advisories, and because of the relatively low cost of chemical analysis for this analyte, EPA recommends that this metal be monitored at all agricultural sites, especially those for which little or no monitoring data are available. Additionally, states may also want to analyze for other metals (arsenic, cadmium, and selenium). States may include any of the recommended EPA target analytes and any additional target analytes in their screening programs when site-specific information on an agricultural watershed suggests that these contaminants may be present at levels of concern for human health.

***Suburban/Urban.*** Water and sediment quality are often regularly monitored in suburban and urban areas, and selection of target analytes should be based on these data when available. Some suburban watersheds of today were agricultural watersheds during the early twentieth century. Arsenic compounds were widely used as pesticides in the early 1900s, as were organochlorine pesticides. These contaminants may still be present in farmland abandoned after the 1940s. As a result of the rapid population growth in recent years, other suburban areas have been built on former industrial sites, so historical information on land use should be obtained by states whenever possible and reviewed carefully during the target analyte selection process.

Several of the organophosphates as well as organochlorine pesticides have had wide use in control of pests around domestic structures as well as in lawn and garden applications (see Table 4-3). Chlorpyrifos and diazinon are currently used by pest control applicators and the general public (Robinson et al., 1994), and diazinon has been reported at high concentrations in effluents from POTWs in some suburban/urban areas (Amato et al., 1992; Burkhard and Jensen, 1993). Historically, chlordane was used extensively in termite control around homes and DDT was used as a general all-purpose insecticide. Nationally, chlordane and DDT are responsible for 3 and 1 percent, respectively, of the advisories currently in effect, and their use within suburban/urban watersheds should be considered as should the use of any of the pesticides registered for use around domestic structures or in lawn and garden applications. Depending on the proximity of some suburban/urban sites to industrial areas, states may also wish to review historic or current information on production sites associated with any of the pesticides, PAHs, PCBs, and dioxin/furans. Because of the historic and current uses of mercury in a variety of industrial processes, because it has triggered issuance of the largest number of U.S. advisories, and because of the relatively low cost of chemical analysis, EPA recommends that this metal be monitored at all suburban/urban sites, especially those where either little or no monitoring data

are available. States should include any of the recommended EPA target analytes and any additional target analytes in their screening programs when site-specific information on a suburban/urban watershed suggests that these contaminants may be present at levels of concern for human health.

***Industrial.*** All of the recommended target analytes can enter waterbodies through releases from industrial processes, Superfund sites, or landfills. Often water and sediment data are available to help guide the selection of the target analytes that should be given high priority with respect to analysis. Selection of analytes for analysis in industrial watersheds should be guided by knowledge of the type of industrial production that has existed in the past or is currently present in the watershed. Historical information is particularly important since potential contaminants may still be present at abandoned industrial sites or contained in sediments in receiving waterbodies. Sources of these target analytes are listed in Section 4.3, which contains the individual target analyte profiles and descriptions of the types of industries that may contribute to releases of these specific pollutants. Again, the states should review all existing water and sediment quality data available before selecting the specific target analytes for analysis at each site. Because of the historic and current uses of mercury in a variety of industrial processes, because it has triggered issuance of the largest number of U.S. advisories, and because of the relatively low cost of chemical analysis, EPA recommends that this metal be monitored at all industrial sites, especially those where little or no monitoring data are available. The other metals, including tributyltin, should also be considered for analysis based on existence of industrial production facilities, waste disposal facilities (e.g., Superfund or hazardous waste sites, and landfills), or shipyards where these target analytes may have been released to the environment. With respect to the pesticides, sites of production, formulation, and packaging facilities can all potentially be sites for release of these contaminants into the surrounding environment. Petroleum refining and coal gasification and processing facilities can also be sites for discharges of PAHs. PCBs can be released from historic landfills where PCB-containing equipment was disposed of or from sites of historic PCB production or use. Dioxins and dibenzofurans are likely to be found in proximity to historic or current industrial sites such as bleached kraft paper mills or production facilities for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), 2,4,5-trichlorophenol (2,4,5-TCP), and/or silvex and medical, municipal, or industrial combustors or incinerators. States should include any of the recommended EPA target analytes and any additional target analytes in their screening programs when site-specific information on an industrial watershed suggests that these contaminants may be present at levels of concern for human health.

Specific factors that have been considered in the selection of the recommended 25 target analytes and sources for their release into the environment are summarized in the next section. Chemical pollutants that are currently under review by EPA's Office of Water for inclusion as recommended target analytes are discussed in Section 4.4.

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## 4.3 TARGET ANALYTE PROFILES

### 4.3.1 Metals

Five metals—arsenic, cadmium, mercury, selenium, and tributyltin—are recommended as target analytes in screening studies. Arsenic, cadmium, and mercury have been included in at least five of the eight major fish contaminant monitoring programs reviewed by the 1993 Workgroup (see Appendix E). It should be noted, however, that with respect to arsenic, all monitoring programs measured total arsenic rather than inorganic arsenic. Selenium was monitored in four national monitoring programs. Tributyltin, a constituent in antifouling paints was not recommended for analysis in any of the national programs evaluated by the 1993 Workgroup. As of 1993, fish consumption advisories were in effect for arsenic, cadmium, mercury, selenium, and tributyltin in 1, 2, 29, 5, and 1 states, respectively (Table 4-2). As of 1998, fish advisories were in effect for arsenic, cadmium, mercury, and selenium in 3, 3, 40, and 11 states, respectively. No states had active advisories for tributyltin (U.S. EPA, 1999c). Also, with the exception of tributyltin, these metals have been identified as having the greatest potential toxicity resulting from ingestion of contaminated fish and shellfish (NAS, 1991).

#### 4.3.1.1 Arsenic—

Arsenic is the twentieth most abundant element in the earth's crust and naturally occurs as a sulfide in a variety of mineral ores containing copper, lead, iron, nickel, cobalt, and other metals (Eisler, 1988; Merck Index, 1989; Woolson, 1975). Arsenic is released naturally to the atmosphere from volcanic eruptions and forest fires (Walsh et al., 1979) and to water via natural weathering processes (U.S. EPA, 1982b). Arsenic also has several major anthropogenic sources including industrial emissions from coal-burning electric generating facilities, releases, as a byproduct of nonferrous metal (gold, silver, copper, lead, uranium, and zinc) mining and smelting operations (Eisler, 1988; May and McKinney, 1981; NAS, 1977), releases associated with its production and use as a wood preservative (primarily as arsenic trioxide), and application as an insecticide, herbicide, algicide, and growth stimulant for plants and animals (Appendix F) (Eisler, 1988). Arsenic releases are also associated with leaching at hazardous waste disposal sites and discharges from sewage treatment facilities. Arsenic trioxide is the arsenic compound of chief commercial importance (U.S. EPA, 1982b) and was produced in the United States until 1985 at the ASARCO smelter near Tacoma, Washington. Arsenic is no longer produced commercially within the United States in any significant quantities, but arsenic compounds are imported into the United States primarily for use in various wood preservative and pesticide formulations.

The toxicity of arsenicals is highly dependent upon the nature of the compounds, and particularly upon the valency state of the arsenic atom (Frost, 1967; Penrose, 1974; Vallee et al., 1960). Typically, compounds containing trivalent (+3) arsenic are much more toxic than those containing pentavalent (+5) arsenic. The valency of the arsenic atom is a more important factor in determining toxicity than the

organic or inorganic nature of the arsenic-containing compound (Edmonds and Francesconi, 1993). With respect to inorganic arsenic compounds, salts of arsenic acid (arsenates) with arsenic in the pentavalent state are less toxic than arsenite compounds with arsenic in the trivalent state (Penrose, 1974). Because some reduction of arsenate (pentavalent arsenic) to arsenite (trivalent arsenic) might occur in the mammalian body (Vahter and Envall, 1983), it would be unwise to disregard the possible toxicity of inorganic arsenic ingested in either valency state (Edmonds and Francesconi, 1993).

Seafood is a major source of trace amounts of arsenic in the human diet. However, arsenic in the edible parts of fish and shellfish is predominantly present as the arsenic-containing organic compound arsenobetaine (Cullen and Reimer, 1989; Edmonds and Francesconi, 1987a; NAS, 1991). Arsenobetaine is a stable compound containing a pentavalent arsenic atom, which has been shown to be metabolically inert and nontoxic in a number of studies (Cannon et al., 1983; Bos et al., 1985; Kaise et al., 1985; Sabbioni et al., 1991; Vahter et al., 1983) and is not generally considered a threat to human health (ATSDR, 1998a). Inorganic arsenic, although a minor component of the total arsenic content of fish and shellfish when compared to arsenobetaine, presents potential toxicity problems. To the degree that inorganic forms of arsenic are either present in seafood or, upon consumption, may be produced as metabolites of organic arsenic compounds in seafood, some human health risk, although small, would be expected (NAS, 1991).

Inorganic arsenic is very toxic to mammals and has been assigned to Toxicity Class I based on oral toxicity tests (U.S. EPA, 1998d). Use of several arsenical pesticides has been discontinued because of the health risks to animals and man. Inorganic arsenic also has been classified as a human carcinogen (A), and long-term effects include dermal hyperkeratosis, dermal melanosis and carcinoma, hepatomegaly, and peripheral neuropathy (IRIS, 1999) (Appendix G).

Total arsenic (inclusive of both inorganic and organic forms) has been included in five of the eight national monitoring programs evaluated by the 1993 Workgroup (Appendix E). Arsenic and arsenic-containing organic compounds have not been shown to bioaccumulate to any great extent in aquatic organisms (NAS, 1977). Experimental evidence indicates that inorganic forms of both pentavalent and trivalent arsenic bioaccumulate minimally in several species of finfish including rainbow trout, bluegill, and fathead minnows (ASTER, 1999). A bioconcentration factor (BCF) value of 350 was reported for the American oyster (*Crassostrea virginica*) exposed to trivalent arsenic (Zaroogian and Hoffman, 1982).

In 1984 and 1985, the U.S. Fish and Wildlife Service collected 315 composite samples of whole fish from 109 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt and Brumbaugh, 1990). The authors reported the the maximum, geometric mean, and 85<sup>th</sup> percentile concentrations for total arsenic were 1.5, 0.14, and 0.27 ppm (wet weight), respectively. No information, however, was available on the percentage of inorganic arsenic in the fish sampled in the NCBP study. Kidwell et al. (1995)

conducted an analysis of total arsenic levels in bottom-feeding and predator fish using the 1984-1985 data from the NCBP study. These authors reported that the mean total arsenic tissue concentrations of  $0.16 \pm 0.23$  ppm in bottom feeders and  $0.16 \pm 0.14$  ppm in predator fish were not significantly different.

Edmonds and Francesconi (1993) summarized existing data from studies conducted outside the United States comparing concentrations of total arsenic, organic arsenic, and inorganic arsenic in marine fish and shellfish. Inorganic arsenic was found to represent from 0 to 44 percent of the total arsenic in marine fish and shellfish species surveyed. Residue concentrations of inorganic arsenic in the tissues typically ranged from 0 to 5.6 ppm (wet weight basis); but were generally less than 0.5 ppm for most species. In a study of six species of freshwater fish monitored as part of the Lower Columbia River study, inorganic arsenic represented from 0.1 to 27 percent of the total arsenic, and tissue residues of inorganic arsenic ranging from 0.001 to 0.047 ppm (wet weight) were 100 times lower than those reported for marine species (Tetra Tech, 1995).

In 1993, only one state (Oregon) had an advisory in effect for arsenic contamination (RTI, 1993). As of 1998, there were three advisories in effect in three states (Louisiana, Oregon, and Washington) for this metal (U.S. EPA, 1999c). Because it is the concentration of inorganic arsenic in fish and shellfish that poses the greatest threat to human health, EPA recommends that total inorganic arsenic (not total arsenic) be analyzed in contaminant monitoring programs. A chemical analysis procedure for determining total inorganic arsenic residues in fish and shellfish tissues is provided in Appendix H. Total inorganic arsenic should be considered for inclusion in state fish and shellfish monitoring programs in areas where it occurs in geologic formations, sites where mining or smelter operations have occurred, or where its use is or has been extensive. States should contact their appropriate state agencies to obtain information on the historic and current uses of arsenic particularly as a wood preservative and in agricultural pesticides.

### 4.3.1.2 Cadmium—

Cadmium is commonly found in zinc, lead, and copper deposits (May and McKinney, 1981). It is released into the environment from several anthropogenic sources: smelting and refining of ores, electroplating, application of phosphate fertilizers, surface mine drainage (Frag et al., 1998; U.S. EPA, 1978), and waste disposal operations (municipal incineration and land application) (U.S. EPA, 1979a, 1987c). Cadmium is also used in the manufacture of paints, alloys, batteries, and plastics and has been used in the control of moles and plant diseases in lawns.

Cadmium is a cumulative human toxicant; it has been shown to cause renal dysfunction and a degenerative bone disease, Itai-Itai, in Japanese populations exposed via consumption of contaminated rice, fish, and water. Because cadmium is retained in the kidney, older individuals (over 40-50 years of age) typically have both the highest renal concentrations of cadmium and the highest prevalence of renal dysfunction (U.S. EPA, 1979a). Cadmium is a known

carcinogen in animals, and there is limited evidence of the carcinogenicity of cadmium or cadmium compounds in humans. It has been classified by EPA as a probable human carcinogen by inhalation (B1) (IRIS, 1999).

Cadmium has been found to bioaccumulate in fish and shellfish tissues in fresh water (Schmitt and Brumbaugh, 1990) and in estuarine/marine waters (NOAA, 1987, 1989a) nationwide. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 315 composite samples of whole fish from 109 stations nationwide as part of the NCBP (Schmitt and Brumbaugh, 1990). The authors reported the maximum, geometric mean, and 85<sup>th</sup> percentile concentrations for cadmium were 0.22, 0.03, and 0.05 ppm (wet weight), respectively. In the NCBP study, geometric mean concentrations of cadmium in freshwater fish were found to have declined from 0.07 ppm in 1976 to 0.03 ppm in 1984 (Schmitt and Brumbaugh, 1990). This trend contradicts the general trend of increasing cadmium concentrations in surface waters, which Smith et al. (1987) attribute to increasing U.S. coal combustion (Schmitt and Brumbaugh, 1990). Kidwell et al. (1995) conducted an analysis of cadmium concentrations in bottom-feeding and predatory fish species using the 1984-1985 data from the NCBP study. These authors found that mean cadmium tissue concentration (whole fish samples) of  $0.04 \pm 0.05$  ppm in bottom feeders (e.g., carp, white sucker, and channel catfish) was significantly higher than the mean cadmium tissue concentration of  $0.01 \pm 0.02$  ppm found in predator fish (e.g., trout, walleye, largemouth bass).

In 1993, only two states (New York and Ohio) had issued fish advisories for cadmium contamination (RTI, 1993). As of 1998, there were seven advisories in effect in three states (Maine, New Jersey, and New York) for this heavy metal (U.S. EPA, 1999c). Two of these states, New York and New Jersey, have issued advisories for this metal in all of their marine coastal waters. Maine has a statewide wildlife advisory in effect for cadmium in moose liver and kidney tissue (U.S. EPA, 1999c). Cadmium should be considered for inclusion in all state fish and shellfish contaminant monitoring programs in areas where it occurs in geologic formations, where mining or smelter operations have occurred, or where its use is or has been extensive.

### 4.3.1.3 Mercury—

A major source of atmospheric mercury is the natural degassing of the earth's crust, amounting to 2,700 to 6,000 tons per year (WHO, 1990). Primary points of entry of mercury into the environment from anthropogenic sources include mining and smelting, industrial processes including chlorine-alkali production facilities and atmospheric deposition resulting from combustion of coal and other fossil fuels and municipal and medical refuse incinerators (U.S. EPA, 1997c; Glass et al., 1990). Primary industrial uses of mercury are in the manufacture of batteries, vapor discharge lamps, rectifiers, fluorescent bulbs, switches, thermometers, and industrial control instruments (May and McKinney, 1981), and these products ultimately end up in landfills or incinerators. Mercury has also been used as a slimicide in the pulp and paper industry, as an antifouling and mildew-proofing

agent in paints, and as an antifungal seed dressing (ATSDR, 1998; *Farm Chemicals Handbook*, 1989; Friberg and Vostal, 1972).

Although mercury use and losses from industrial processes in the United States have been reduced significantly since the 1970s, mercury contamination associated with increased fossil fuel combustion is of concern in some areas and may pose more widespread contamination problems in the future. An estimated 5,000 tons of mercury per year is released into the environment from fossil fuel burning (Klaassen et al., 1986). The best estimate of annual anthropogenic U.S. emissions of mercury in 1994-1995 was 158 tons. Of this, about 87 percent was released from combustion sources, including waste and fuel combustion. (U.S. EPA, 1997). There is also increasing evidence of elevated mercury concentrations in areas where acid rain is believed to be a factor (NESCAUM, 1998; Sheffy, 1987; Wiener, 1987). Volatilization from surfaces painted with mercury-containing paints, both indoors and outdoors, may have been a significant source in the past (Agocs et al., 1990; Sheffy, 1987). The United States estimated that 480,000 pounds of mercuric fungicides were used in paints and coatings in 1987 (NPCA, 1988). In July 1990, EPA announced an agreement with the National Paint and Coatings Association to cancel all registrations for use of mercury or mercury compounds in interior paints and coatings. In May 1991, the paint industry voluntarily canceled all remaining registrations for mercury in exterior paints.

Cycling of mercury in the environment is facilitated by the volatile character of its metallic form and by bacterial transformation of metallic and inorganic forms to stable alkyl mercury compounds, particularly in bottom sediments, which leads to bioaccumulation of mercury (Wood, 1974). Practically all mercury in fish tissue is in the form of methylmercury (Bache et al., 1971; Bloom, 1992; Kannan et al., 1998; Spry and Wiener, 1991), which is toxic to humans (NAS, 1991; Tollefson, 1989), with the percentage of methylmercury to total mercury in the muscle tissue increasing as the fish ages (Bache et al., 1971). Several studies have shown that mercury concentrations in fish tissue generally increase with age, and therefore size (length or weight), owing to methylmercury accumulation with increasing duration of exposure (Driscoll et al., 1994; Jackson, 1990; Johnson, 1987; Lange et al., 1993); however this relationship is not as strongly correlated in all environmental situations or for all fish species (Goldstein et al., 1996; Neumann et al., 1997).

EPA has classified methylmercury as a Group C, possible human carcinogen, based on inadequate data in humans and limited evidence in animals (Appendix G). No persuasive evidence of increased carcinogenicity attributable to methylmercury exposure was observed in three human studies; however, interpretation of these studies was limited by poor study design and other problems. Animal studies have shown significant increases in the incidences of kidney tumors in male, but not in female, mice (IRIS, 1999).

Both inorganic and organic forms of mercury are neurotoxicants. Fetuses exposed to organic mercury have been found to be born mentally retarded and

with symptoms similar to those of cerebral palsy (Marsh, 1987; U.S. EPA, 1997c). Individuals exposed to mercury via long-term ingestion of mercury-contaminated fish have been found to exhibit a wide range of symptoms, including numbness of the extremities, tremors, spasms, personality and behavior changes, difficulty in walking, deafness, blindness, and death (U.S. EPA, 1997c). Organomercury compounds were the causative agents of Minamata Disease, a neurological disorder reported in Japan during the 1950s among individuals consuming contaminated fish and shellfish (Kurland et al., 1960), with infants exposed prenatally found to be at significantly higher risk than adults. Another methylmercury poisoning incident involving fish and shellfish occurred in 1965 in Niigata, Japan. A third methylmercury poisoning incident occurred in the late 1960s and early 1970s in Iraq; however, this last incident was associated with the accidental consumption of seed grain treated with organomercury fungicide (U.S. EPA, 1997c). The EPA is especially concerned about evidence that the fetus is at increased risk of adverse neurological effects from exposure to methylmercury (e.g., Marsh et al., 1987; Piotrowski and Inskip, 1981; Skerfving, 1988; WHO, 1976, 1990; U.S. EPA, 1997c).

The EPA has set an interim Reference Dose (RfD) for methylmercury of  $0.1 \mu\text{g}/\text{kg}\text{-d}$  (IRIS 1999). The National Academy of Sciences (NAS) conducted an independent assessment of the interim RfD. They concluded "On the basis of its evaluation, the committee's consensus is that the value of EPA's current RfD for methylmercury,  $0.1 \mu\text{g}/\text{kg}$  per day, is a scientifically justifiable level for the protection of public health". However, the NAS recommended that the Iraqi study no longer be used as the scientific basis for the RfD. In addition, the NAS recommended that the developmental neurotoxic effects of methylmercury reported in the Faroe Islands study should be used as the basis for the derivation of the RfD." (NAS, 2000)

Mercury has been found in both fish and shellfish from estuarine/marine (NOAA, 1987, 1989a) and fresh waters (Schmitt and Brumbaugh, 1990) at diverse locations nationwide. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 315 composite samples of whole fish from 109 stations nationwide as part of the National Contaminant Biomonitoring Program (NCBP) (Schmitt and Brumbaugh, 1990). The authors reported that the maximum, geometric mean, and 85<sup>th</sup> percentile concentrations for mercury were 0.37, 0.10, and 0.17 ppm (wet weight), respectively. In contrast to cadmium and selenium, concentrations of mercury in freshwater fish tissue did not decline between 1976 and 1984 (Schmitt and Brumbaugh, 1990). Kidwell et al. (1995) conducted an analysis of mercury levels in bottom-feeding and predator fish using the 1984-1985 data from the NCBP study. These authors reported that the mean mercury tissue concentration (whole fish samples) of  $0.12 \pm 0.08$  ppm in predator fish (e.g., trout, walleye, largemouth bass) was significantly higher than the mean tissue concentration of  $0.08 \pm 0.006$  ppm in bottom feeders (e.g., carp, white sucker, and channel catfish).

Mercury, the only metal analyzed as part of the EPA National Study of Chemical Residues in Fish, was detected at 92 percent of 374 sites surveyed. Maximum,

arithmetic mean, and median concentrations in fish tissue were 1.77, 0.26, and 0.17 ppm (wet weight), respectively (U.S. EPA, 1991h, 1992c, 1992d). Bahnick et al. (1994) analyzed the NSCRF data by fish species and reported that mean mercury concentrations in bottom feeders (whole body samples) were generally lower than concentrations for predator fish (fillet samples). Carp, white sucker, and channel catfish (bottom feeders) had average tissue concentrations of 0.11, 0.11, and 0.09 ppm, respectively. Largemouth bass, smallmouth bass, and walleye (predator species) had average tissue concentrations of 0.46, 0.34, and 0.52 ppm, respectively (Bahnick et al., 1994). With regard to the source of the mercury contamination, Bahnick et al. (1994) reported that the highest mean concentration of mercury was detected in fish sampled near public treatment works (0.59 ppm); however, background sites and sites near wood preserving facilities exhibited the second (0.34 ppm) and third (0.31 ppm) highest mean mercury concentrations. The authors also reported that most of the higher tissue concentrations of mercury were detected in freshwater fish samples collected in the Northeast.

Recently, the northeastern states and eastern Canadian provinces issued their own mercury study, including a comprehensive analysis of mercury concentrations in a variety of freshwater sportfish (NESCAUM, 1998). This study involved a large number of sampling sites, including remote lake sites that did not receive point source discharges. Top-level piscivores (i.e., predator fish), such as walleye, chain pickerel, and large and smallmouth bass, were typically found to exhibit the highest concentrations, with mean tissue residues greater than 0.5 ppm and maximum residues exceeding 2 ppm. One largemouth bass sample was found to contain 8.94 ppm of mercury, while a smallmouth bass sampled contained 5 ppm. A summary of the range and the mean concentrations found in eight species of sportfish sampled is shown in Table 4-4 (NESCAUM, 1998).

**Table 4-4. Total Mercury and Methylmercury Concentrations in Estuarine Fish from South Florida**

<b>Species</b>	<b>Mean mercury concentration<sup>a</sup> (ppm) and range</b>	<b>Mean methylmercury<sup>a</sup> concentration (ppm) and range</b>
Hardhead catfish	1.94 (0.44-4.64)	1.54 (0.18-4.42)
Gafftopsail catfish	3.0 (0.76-10.10)	1.86 (0.72-4.50)
Sand seatrout	2.41 (2.21-2.61)	2.04 (1.60-2.47)
Sand seaperch	0.48 (0.40-0.54)	0.42 (0.40-0.49)
Pinfish	0.54 (0.32-1.06)	0.44 (0.20-0.90)
White grunt	0.49 (0.28-1.03)	0.49 (0.31-0.99)
Lane snapper	0.57 (0.22-1.03)	0.58 (0.19-1.27)
Spot	0.29 (0.11-0.43)	0.24 (0.06-0.40)

<sup>a</sup>Concentrations are in ppm ( $\mu\text{g/g}$ ) wet weight basis.

Source: Kannan et al., 1998.

EPA's Office of Water also recently published results of a national survey of mercury concentrations in fish (U.S. EPA, 1999d). This survey compiled state data on tissue residue levels of mercury in fish analyzed by 39 states between 1990 and 1995. The range of mean mercury concentrations (ppm) for the nine major fish species reported were as follows: largemouth bass, 0.001-8.94; smallmouth bass, 0.008-3.34; walleye, 0.008-3.0; northern pike, 0.10-4.4; channel catfish, 0.001-2.57; bluegill sunfish, 0.001-1.68; common carp, 0.001-1.8; white sucker, 0.002-1.71; and yellow perch, 0.01-2.14. All mercury concentrations used in the study were expressed on a wet weight and fillet basis. While the majority of the finfish sampled were freshwater species, some estuarine and marine species were also included; however, the report excluded all nonfish species such as turtles, molluscs, and crustaceans. Although comparison of data between states was difficult because of differences in sampling strategies (representative versus targeted), differences in analytical procedures, and the fact that mercury concentrations may vary with age of the fish, the analysis did indicate that both the magnitude and variability of mercury concentrations were greater in higher trophic level fish species.

Another recent study was conducted to assess total mercury and methylmercury concentrations in estuarine fish from south Florida coastal waters (Kannan et al., 1998). The authors reported that concentrations of total mercury in fish muscle tissue ranged between 0.03 and 2.22 ppm (mean: 0.31 ppm) (wet weight basis), with methylmercury contributing 83 percent of the total mercury. The mean concentrations and range of total mercury and methylmercury in muscle tissue of different species collected from south Florida's coastal waters are shown in Table 4-4.

In another study, methylmercury concentrations in muscle tissue of nine species of sharks were analyzed from four different locations along the coast of Florida (Hueter et al., 1995). Muscle tissue methylmercury concentrations averaged 0.88 ppm (wet weight) and ranged from 0.06 to 2.87 ppm, with 31 percent of the samples tested exceeding 1 ppm. A positive correlation was found between methylmercury concentration and the body length (size) of the shark, such that sharks larger than 2 m in total length contained methylmercury concentrations >1 ppm. Sharks collected off the southern and southwestern coastal areas contained significantly higher concentrations than those caught in the northeast coastal region (Cape Canaveral and north). Methylmercury concentrations were highest in the Caribbean reef shark (*Carcharhinus perezii*). The two most abundant shark species in the U.S. East Coast commercial shark fishery, the sandbar (*C. plumbeus*) and blacktip (*C. limbatus*) sharks, are of special public health concern. Although the mean methylmercury concentration in the sandbar shark (0.77 ppm) was below the average for all sharks, sandbar shark tissues contained up to 2.87 ppm methylmercury, and 20.9 percent of the sampled fish exceeded 1 ppm. Of more concern is that 71.4 percent of the blacktip shark samples (mean, 1.3 ppm) exceeded 1 ppm methylmercury. The authors suggest that continued monitoring of methylmercury concentrations in various shark species is warranted, since these fish are taken in both recreational and commercial fisheries. Similarly, on the West Coast, Fairey et al. (1997) reported

that the highest concentrations of mercury found in all of the fish species sampled as part of a fish monitoring effort in the San Francisco Bay and Estuary were detected in leopard shark muscle tissue (1.26 ppm wet weight basis).

In 1993, 898 fish advisories had been issued in 29 states as a result of mercury contamination (see Figure 4-1). In particular, mercury was included in a large number of the fish advisories in effect for lakes in Minnesota, Wisconsin, and Michigan and for rivers and lakes in Florida (RTI, 1993). As of 1998, 1,931 advisories had been issued in 40 states for this metal, and mercury is responsible for more than 68 percent of all fish advisories issued in the United States. In addition, 10 states have statewide advisories in effect for mercury in freshwater lakes and/or rivers and 5 Gulf Coast states have statewide mercury advisories in effect for their coastal marine waters (U.S. EPA, 1999c).

Because of its widespread occurrence in fish across the United States, mercury should be monitored in all state fish and shellfish contaminant monitoring programs at all stations. Only one national program reviewed by the 1993 Workgroup—EPA 301(h) monitoring program—recommended analyzing specifically for methylmercury; however, six programs recommended analyzing for total mercury (Appendix E). Because of the higher cost of methylmercury analysis two to three times greater than for total mercury analysis). EPA recommends that total mercury be determined in state fish contaminant monitoring programs and the conservative assumption be made that all mercury is present as methylmercury so as to be most protective of human health. It should be noted that Bache et al. (1971) analyzed methylmercury concentrations in lake trout of known ages and found that methylmercury concentration and the ratio of methylmercury to total mercury increased with age. Relative proportions of methylmercury in fish varied between 30 and 100 percent, with methylmercury concentrations lower than 80 percent occurring in fish 3 years of age or younger. Thus, when high concentrations of total mercury are detected, and if resources are sufficient, states may wish to repeat sampling and obtain more specific information on actual concentrations of methylmercury in various age or size classes of fish.

### 4.3.1.4 Selenium—

Selenium is a natural component of many soils, particularly in the west and southwest regions of the United States (NAS, 1991). It enters the environment primarily via emissions from oil and coal combustion (May and McKinney, 1981; Pillay et al., 1969). Selenium is an essential nutrient but is toxic to both humans and animals at high concentrations (NAS, 1991). Long-term adverse effects from ingestion by humans have not been studied thoroughly. EPA has determined that the evidence of carcinogenicity of selenium in both humans and animals is inadequate and, therefore, has assigned this metal a D carcinogenicity classification (IRIS, 1999).

Selenium is frequently detected in ground and surface waters in most regions of the United States and has been detected in marine fish and shellfish (NOAA,

4. TARGET ANALYTES

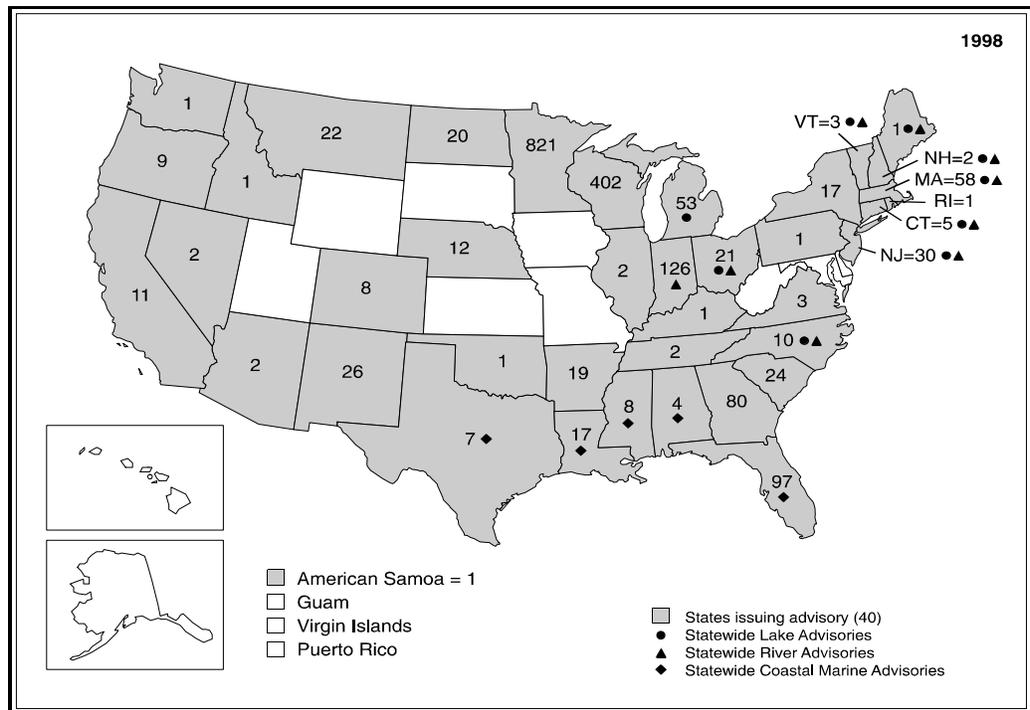
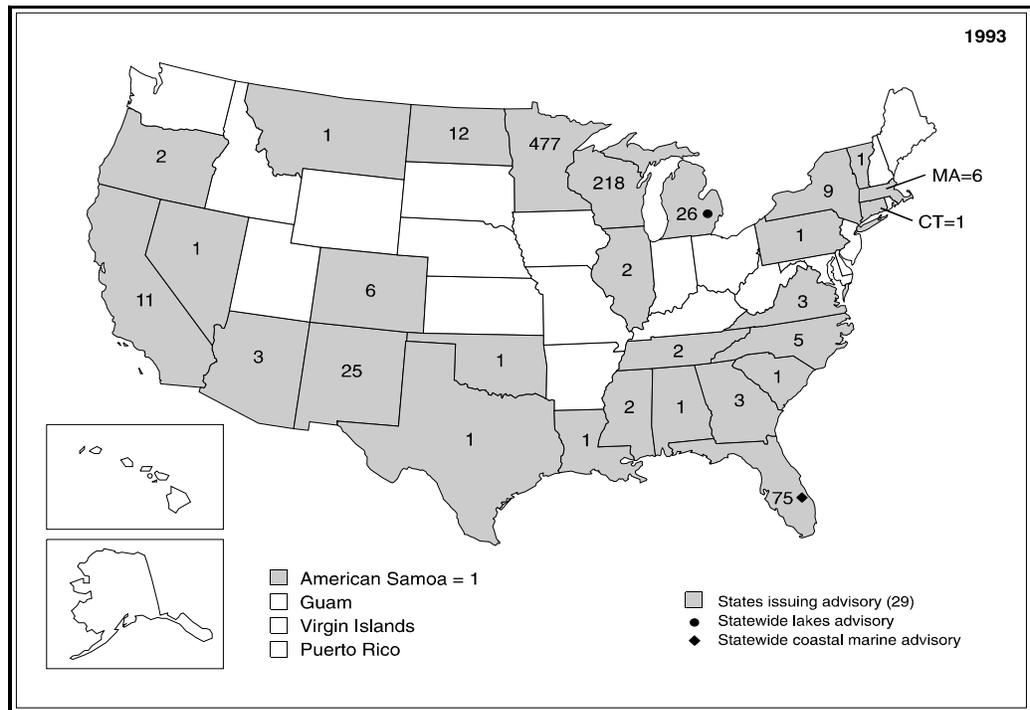


Figure 4-1. States issuing fish and shellfish advisories for mercury.

1987, 1989a) and in freshwater fish (Schmitt and Brumbaugh, 1990) from several areas nationwide. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 315 composite samples of whole fish from 109 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt and Brumbaugh, 1990). The authors reported the maximum, geometric mean, and 85<sup>th</sup> percentile concentrations for selenium were 2.30, 0.42, and 0.73 ppm (wet weight), respectively. Kidwell et al. (1995) conducted an analysis of selenium concentrations in bottom-feeding and predator fish using the 1984-1985 data from the NCBP study. Mean selenium tissue concentrations (whole fish samples) were not significantly different in bottom feeders ( $0.50 \pm 0.41$  ppm) as compared to predator fish ( $0.50 \pm 0.42$  ppm). Like cadmium, concentrations of selenium declined in fish tissues between 1976 and 1984 (Schmitt and Brumbaugh, 1990).

In a more recent study (May 1993 to January 1994), selenium concentrations in the tissues of fish from the Pigeon River and Pigeon Lake in Michigan were examined. Mean selenium concentrations in white sucker fillets were  $0.49 \pm 0.19$ ,  $1.8 \pm 0.96$ , and  $1.7 \pm 0.80$  ppm (wet weight) in samples taken from the Upper Pigeon River, Lower Pigeon River, and Pigeon Lake, respectively. At these same locations, northern pike fillets contained selenium concentrations of  $0.88 \pm 0.22$ ,  $1.1 \pm 0.91$ , and  $2.2 \pm 0.90$  ppm (wet weight), respectively (Besser et al., 1996). This study was conducted to assess the potential hazard of selenium leaching from a coal fly ash disposal area.

Selenium was monitored in four national fish contaminant monitoring programs reviewed by the EPA 1993 Workgroup (Appendix E). Definitive information concerning the chemical forms of selenium found in fish and shellfish is not available (NAS, 1976, 1991).

In 1993, five states (California, Colorado, North Carolina, Texas, and Utah) had issued advisories for selenium contamination in fish (RTI, 1993). As of 1998, there were 11 advisories in effect in these same five states for this heavy metal (U.S. EPA, 1999c). These advisories include one wildlife advisory in Nevada for selenium in several species of waterfowl. Selenium should be considered for inclusion in all state fish and shellfish monitoring programs in areas where it occurs in geologic formations (particularly in the western and southwestern states) and near sites where oil or coal combustion currently occurs or historically has occurred.

### 4.3.1.5 Tributyltin Compounds—

Tributyltin compounds belong to the organometallic family of tin compounds that have been used as biocides, disinfectants, and antifoulants. Antifoulant paints containing tributyltin compounds were first registered for use in the United States in the early 1960s (Appendix F). Tributyltin compounds are used in paints applied to boat and ship hulls as well as to crab pots, fishing nets, and buoys to retard the growth of fouling organisms. These compounds were also registered for use as wood preservatives, disinfectants, and biocides in cooling towers, pulp and paper mills, breweries, leather processing facilities, and textile mills (U.S. EPA, 1988c).

Tributyltin compounds are acutely toxic to aquatic organisms at concentrations below 1 ppb and are chronically toxic to aquatic organisms at concentrations as low as 0.002 ppb (U.S. EPA, 1988c). EPA initiated a Special Review of tributyltin compounds used as antifoulants in January of 1986 based on concerns over its adverse effects on nontarget aquatic species. Shortly thereafter the Organotin Antifouling Paint Control Act (OAPCA) was enacted in June 1988, which contained interim and permanent tributyltin use restrictions as well as environmental monitoring, research, and reporting requirements. The Act established interim release rate restrictions under which only tributyltin-containing products that do not exceed an average daily release rate of 4 micrograms organotin/cm<sup>2</sup>-d can be sold or used. The OAPCA also contained a permanent provision to prohibit the application of tributyltin antifouling paints to non-aluminum vessels under 25 meters (82 feet) long (U.S. EPA, 1988c).

Tributyltin oxide appears to be toxic to animals, with oral LD<sub>50</sub>s ranging between 52 and 194 mg/kg (ATSDR, 1992; HSDB, 1999; WHO, 1999). Immunotoxicity is the critical effect produced by chronic exposure to tributyltin. Insufficient data are available to evaluate the carcinogenicity of tributyltin oxide compounds; therefore, EPA has listed this compound in Group D (Appendix G) (IRIS, 1999).

Tributyltins have been found to bioaccumulate in fish, bivalve mollusks, and crustaceans. Bioconcentration factors have been reported to range from 200 to 4,300 for finfish, from 2,000 to 6,000 for bivalves, and a BCF value of 4,400 was reported for crustaceans (U.S. EPA, 1988c). Tributyltin used to control marine fouling organisms in an aquaculture rearing pen has been found to bioaccumulate in fish tissue (Short and Thrower, 1987a and 1987b). Tsuda et al. (1988) reported a BCF value of 501 for tributyltin in carp (*Cyprinus carpio*) muscle tissue. Martin et al. (1989) reported a similar BCF value of 406 for tributyltin in rainbow trout (*Salmo gairdneri*) and Ward et al. (1981) reported a BCF value of 520 for the sheepshead minnow (*Cyprinodon variegatus*). In an environmental monitoring study conducted in England, a BCF value of 1,000 was reported for tributyltin in seed oysters (*Crassostrea gigas*) (Ebdon et al., 1989).

Tributyltin was not monitored in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). In 1993, only one state, Oregon, had an advisory in effect for tributyltin contamination in shellfish (RTI, 1993). As of 1998, there were no active fish advisories in effect for tributyltin, since the advisory in Oregon was rescinded (U.S. EPA, 1999c).

Tributyltin compounds should be considered for inclusion in all state fish and shellfish contaminant monitoring programs, particularly in states with coastal waters, states bordering the Great Lakes, or states with large rivers where large ocean-going vessels are used for commerce. Tributyltin concentrations have been reported to be highest in areas of heavy boating and shipping activities including shipyards, drydocks, and marinas where tributyltin-containing antifouling paints are often removed and reapplied. Before recoating, old paint containing tributyltin residues is scraped from the vessel hull and these paint scrapings are sometimes washed into the water adjacent to the boat or shipyard despite the tributyltin label

prohibiting this practice (U.S. EPA, 1988c). Tributyltin should be considered for inclusion in state fish and shellfish monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of tributyltin, particularly with respect to its uses in antifouling paints and wood preservatives.

### 4.3.2 Organochlorine Pesticides

The following organochlorine pesticides and metabolites are recommended as target analytes in screening studies: total chlordane (sum of cis- and trans-chlordane, cis- and trans-nonachlor, and oxychlordane), total DDT (sum of 2,4'- and 4,4'-homologues of DDT, DDD, and DDE), dicofol, dieldrin, endosulfan I and II, endrin, heptachlor epoxide, hexachlorobenzene, lindane ( $\gamma$ -hexachlorocyclohexane), mirex, and toxaphene (see Appendix F). Mirex is of particular concern in the Great Lakes states and the southeast states (NAS, 1991). All of these compounds are neurotoxins and most are known or suspected human carcinogens (IRIS, 1999; Sax, 1984).

With the exception of endosulfan I and II, dicofol, and total DDT, each of the pesticides on the recommended target analyte list (Table 4-1) had been included in at least four major fish contaminant monitoring programs (Appendix E), and seven of the compounds had triggered at least one state fish consumption advisory in 1993 (Table 4-2). Although use of some of these pesticides has been terminated or suspended within the United States for over 25 years (Appendix F), these compounds still require long-term monitoring. Many of the organochlorine pesticides that are now banned were used in large quantities for over a decade and are still present in sediments at high concentrations. These organochlorine pesticides are not easily degraded or metabolized and, therefore, persist in the environment. These compounds are either insoluble or have relatively low solubility in water, but are quite lipid-soluble. Because these compounds are not readily metabolized or excreted from the body and are readily stored in fatty tissues, they can bioaccumulate to high concentrations through aquatic food chains to secondary consumers (e.g., fish, piscivorous birds, and mammals including humans).

Pesticides may enter aquatic ecosystems from point source industrial discharges or from nonpoint sources such as aerial drift and/or runoff from agricultural use areas, leaching from landfills, or accidental spills or releases. Agricultural runoff from crop and grazing lands is considered to be the major source of pesticides in water, with industrial waste (effluents) from pesticide manufacturing the next most common source (Li, 1975). Significant atmospheric transport of pesticides to aquatic ecosystems can also result from aerial drift of pesticides, volatilization from applications in terrestrial environments, and wind erosion of treated soil (Li, 1975). Once in water, pesticide residues may become adsorbed to suspended material, deposited in bottom sediment, or absorbed by organisms in which they are detoxified and eliminated or accumulated (Nimmo, 1985).

The reader should note that three of the organochlorine pesticides still have active registrations: endosulfan, lindane, and dicofol. These pesticides are much less persistent in the environment and have a lower bioaccumulation potential than the banned organochlorines. However, agricultural runoff particularly during the period immediately after field application could result in significant levels of these pesticides in fish and shellfish tissues. States should contact their appropriate state agencies to obtain information on both the historic and current uses of these pesticides.

#### 4.3.2.1 Chlordane (Total)—

Chlordane is a multipurpose insecticide that has been used extensively in home and agricultural applications in the United States for the control of termites and many other insects (Appendix F). This pesticide is similar in chemical structure to dieldrin, although less toxic (Toxicity Class II), and has been classified as a probable human carcinogen (B2) by EPA (Appendix G) (IRIS, 1999; Worthing, 1991).

Although the last labeled use of chlordane as a termiticide was phased out in the United States beginning in 1975, it has been monitored in seven national fish contaminant programs evaluated by the EPA 1993 Workgroup (Appendix E) and has been widely detected in freshwater fish (Schmitt et al., 1990) and in both estuarine/marine finfish (NOAA, 1987) and marine bivalves (NOAA, 1989a) at concentrations of human health concern. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). These authors reported the maximum and geometric mean concentrations for the five major degradation products of chlordane (*cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane) were 0.66 and 0.03 ppm, 0.35 and 0.02 ppm, 0.45 and 0.02 ppm, 1.00 and 0.30 ppm, and 0.29 and 0.01 ppm (wet weight), respectively. Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on the major constituents of chlordane (including *cis*- and *trans*-chlordane, *cis*- and *trans*-nonachlor, and oxychlordane) in bottom-feeding and predator fish species. The authors reported there was no significant difference in residues in these two trophic groups of fish except for concentrations of *trans*-chlordane, which were significantly higher in the tissues of bottom feeders. Mean tissue concentrations of *cis*- and *trans*-chlordane, *cis*- and *trans*-nonachlor, and oxychlordane were  $0.03 \pm 0.06$ ,  $0.02 \pm 0.04$ ,  $0.02 \pm 0.04$ ,  $0.03 \pm 0.01$ , and  $0.01 \pm 0.02$  ppm, respectively, for bottom feeders as compared to  $0.02 \pm 0.04$ ,  $0.01 \pm 0.02$ ,  $0.02 \pm 0.03$ ,  $0.03 \pm 0.06$ , and  $0.01 \pm 0.01$  ppm, respectively, for predator species (Kidwell et al., 1995).

The *cis*- and *trans*-isomers of chlordane and *cis*- and *trans*-isomers of nonachlor, which are primary constituents of technical-grade chlordane, and oxychlordane, the major metabolite of chlordane, were also monitored as part of the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). These compounds were detected in fish tissue at the following percentage of the 362

sites surveyed: *cis*-chlordane (64 percent), *trans*-chlordane (61 percent), *cis*-nonachlor (35 percent), *trans*-nonachlor (77 percent), and oxychlordane (27 percent) (U.S. EPA, 1992c, 1992d). The maximum, arithmetic mean, and median concentrations (wet weight) of *cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *trans*-nonachlor, and oxychlordane are summarized in Table 4-5. Mean total chlordane residues from the NSCRF study were highest in bottom feeders such as carp (0.067 ppm), white sucker (0.018 ppm), and channel catfish (0.054 ppm) as compared to predator fish such as largemouth bass (0.029 ppm), smallmouth bass (0.004 ppb), and walleye (0.004 ppm) (Kuehl et al., 1994).

**Table 4-5. Chlordane Constituent Concentrations<sup>a</sup> Detected in the EPA National Study of Chemical Residues in Fish**

Chlordane constituent or metabolite	Maximum	Arithmetic mean	Median
<i>cis</i> -Chlordane	0.378	0.021	0.004
<i>trans</i> -Chlordane	0.310	0.017	0.003
<i>cis</i> -Nonachlor	0.127	0.009	ND
<i>trans</i> -Nonachlor	0.477	0.031	0.009
Oxychlordane	0.243	0.005	ND

ND = Not detected.

<sup>a</sup>Concentrations are in ppm (micrograms/g) on a wet weight basis.

Source: U.S. EPA, 1992c, 1992d.

In 1993, 120 fish advisories in 24 states had been issued as a result of chlordane contamination (see Figure 4-2). As of 1998, there were 104 advisories in effect in 22 states for this pesticide, and New York currently has a statewide advisory for chlordane in all waterfowl (U.S. EPA, 1999c). Because of its extensive use in termite control and its widespread detection in fish tissues, total chlordane (i.e., sum of *cis*- and *trans*-chlordane, *cis*- and *trans*-nonachlor, and oxychlordane) should be considered for inclusion in all state fish and shellfish contaminant monitoring programs (NAS, 1991). Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where chlordane was used historically. In suburban/urban watersheds, the degree of historic use of chlordane as a termiticide around domestic structures should also be evaluated. Sites in industrial watersheds should be reviewed to identify historic sites of chlordane production, formulation, or packaging facilities.

#### 4.3.2.2 DDT (Total)—

Although the use of DDT was terminated in the United States in 1972, DDT and its DDE and DDD metabolites persist in the environment and are known to bioaccumulate (Ware, 1978). DDT, DDD, and DDE have all been classified by EPA as probable human carcinogens (B2) (Appendix G) (IRIS, 1999).

4. TARGET ANALYTES

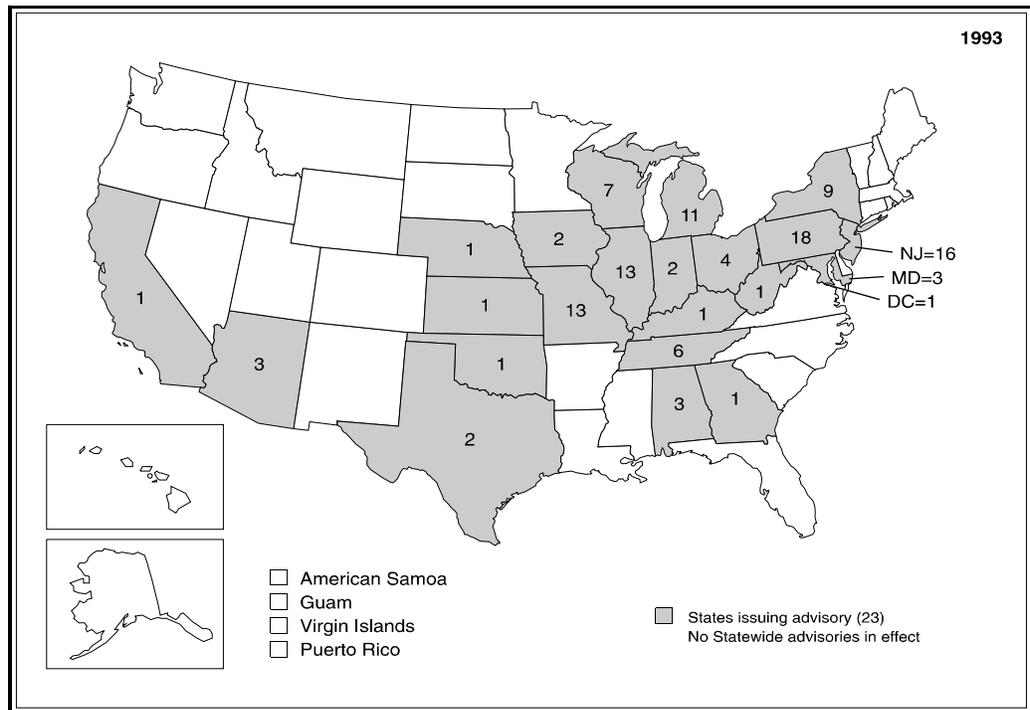


Figure 4-2. States issuing fish and shellfish advisories for chlordane.

DDT or its metabolites have been included as target analytes in as many as seven major fish and shellfish monitoring programs (Appendix E) and contamination has been found to be widespread (NOAA, 1987, 1989a; Schmitt et al., 1990). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Maximum and geometric mean tissue concentrations of DDT, DDE, and DDD in 1984 were 1.79 and 0.03 ppm, 4.74 and 0.19 ppm, and 2.55 and 0.06 ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on DDT and its major metabolites (DDE and DDD) in bottom-feeding and predator fish. The authors reported that there was no significant difference in residues in these two trophic groups of fish. Mean tissue concentrations of DDT, DDE, and DDD were  $0.03 \pm 0.14$ ,  $0.21 \pm 0.46$ , and  $0.07 \pm 0.21$  ppm for bottom feeders as compared to  $0.03 \pm 0.06$ ,  $0.24 \pm 0.55$ , and  $0.06 \pm 0.14$  ppm for predator species, respectively. DDE, the only DDT metabolite surveyed in fish tissue in the EPA National Study of Chemical Residues in Fish, was detected at more sites than any other single chemical pollutant (99 percent of the 362 sites sampled) (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median concentrations of DDE were 14, 0.295, and 0.058 ppm (wet weight), respectively. Mean DDE residues from the NSCRF study were highest in bottom feeders such as carp (0.42 ppm), white sucker (0.08 ppm), and channel catfish (0.63 ppm) as compared to predator species such as largemouth bass (0.06 ppm), smallmouth bass (0.03 ppb), and walleye (0.03 ppm) (Kuehl et al., 1994). In 1993, eight states (Alabama, Arizona, California, Delaware, Massachusetts, Nebraska, New York, and Texas) and the territory of American Samoa had fish consumption advisories in effect for DDT or its metabolites (RTI, 1993). As of 1998, there were 34 advisories in effect in 11 states and the territory of American Samoa for DDT and/or one of its metabolites, DDE or DDD (U.S. EPA, 1999c). In addition, New York has a statewide DDT advisory in effect for mergansers. Because of the extensive national use of this compound and its widespread detection in fish tissues, total DDT (i.e., sum of the 4,4'- and 2,4'-homologues of DDT and of its metabolites, DDE and DDD) should be considered for inclusion in all state fish and shellfish contaminant monitoring programs. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where DDT was applied historically. In suburban/urban watersheds, the degree of historic use of DDT in domestic home and garden applications should be evaluated. Sites in industrial watersheds should be reviewed to identify historic sites of DDT production, formulation, or packaging facilities.

### 4.3.2.3 Dicofol—

Dicofol, one of the three organochlorine target analytes with an active registration, is a miticide/pesticide that was first registered for use in 1957. Currently, dicofol is used primarily on cotton, apples, and citrus crops, mostly in California and Florida (U.S. EPA, 1998c). Dicofol is considered a DDT analog based on its structure and activity (Hayes and Laws, 1991). In the past, dicofol often contained 9 to 15 percent DDT and its analogs. In 1989, EPA required that these contaminants constitute less than 0.1 percent of dicofol (HSDB, 1993).

Historically, dicofol has been used to control mites on cotton and citrus (60 percent), on apples (10 percent), on ornamental plants and turf (10 percent), and on a variety of other agricultural products (20 percent) including pears, apricots, and cherries (*Farm Chemical Handbook*, 1989), as a seed crop soil treatment, on vegetables (e.g., beans and corn), and on shade trees (U.S. EPA, 1992c, 1992d).

Dicofol is moderately toxic to laboratory rats and has been assigned to EPA Toxicity Class III based on an oral LD<sub>50</sub> of 587 mg/kg in rats (U.S. EPA, 1998d) (Appendix F). Technical-grade dicofol induced hepatocellular (liver) carcinomas in male mice; however, results were negative in female mice and in rats (NCI, 1978) and in a second 2-year feeding study in both sexes of rats (U.S. EPA, 1998d). EPA has classified dicofol as a possible human carcinogen (C) (Appendix G) (U.S. EPA, 1998c).

Dicofol was recommended for monitoring by the EPA Office of Water as part of the Assessment and Control of Bioconcentratable Contaminants in Surface Waters Program and has been included in two other national monitoring programs (see Appendix E). Experimental evidence indicates this compound bioaccumulates extensively in bluegill sunfish (BCF from 6,600 to 17,000) (U.S. EPA, 1993a).

In the EPA National Study of Chemical Residues in Fish, dicofol was detected at 16 percent of the 374 sites monitored (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median dicofol concentrations (wet weight basis) were 0.074 ppm, 0.001 ppm, and ND (not detectable). Dicofol concentrations were greater than the quantification limit (0.0025 ppm) in samples from only 7 percent of the sites. Most of the sites where dicofol was detected were in agricultural areas where citrus and other fruits and vegetables are grown (U.S. EPA, 1992c, 1992d). It should be noted that this national study did not specifically target agricultural sites where this pesticide historically had been or currently was used. Dicofol residues in fish could be much higher if sampling were targeted for pesticide runoff, particularly during the period immediately after field application. Mean dicofol residues from the NSCRF study were highest in bottom feeders such as carp (0.88 ppm), white sucker (0.48 ppm), and channel catfish (0.59 ppm) as compared to predator species such as largemouth bass (0.20 ppm), smallmouth bass (not detected), and walleye (not detected) (Kuehl et al., 1994).

In 1993, however, no consumption advisories were in effect for dicofol (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Dicofol should be considered for inclusion in state fish and shellfish contaminant monitoring programs, in areas where its use is or has been extensive. States should contact their appropriate state agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where dicofol is currently used and was used historically. Sites in industrial watersheds should be reviewed to identify historic and current sites of dicofol production, formulation, or packaging facilities.

#### 4.3.2.4 Dieldrin—

Dieldrin is a chlorinated cyclodiene that was widely used in the United States from 1950 to 1974 as a broad spectrum pesticide, primarily on termites and other soil-dwelling insects and on cotton, corn, and citrus crops. Because the toxicity of this persistent pesticide posed an imminent danger to human health, EPA banned the production and most major uses of dieldrin in 1974, and, in 1987, all uses of dieldrin were voluntarily canceled by industry (see Appendix F).

Dieldrin has been classified by EPA as a probable human carcinogen (B2) (Appendix G) (IRIS, 1999) and has been identified as a human neurotoxin (ATSDR, 1991). Dieldrin has been included in seven national monitoring programs (Appendix E) and has been detected nationwide in freshwater finfish (Schmitt et al., 1990) and estuarine/marine finfish and shellfish (NOAA, 1987, 1989a). Because it is a metabolite of aldrin, the environmental concentrations of dieldrin are a cumulative result of the historic use of both aldrin and dieldrin (Schmitt et al., 1990).

In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program. Maximum and geometric mean tissue concentrations of dieldrin in 1984 were 1.39 and 0.04 ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on dieldrin in bottom-feeding and predator fish. These authors reported there was no significant difference in residues in these two trophic groups of fish. Mean tissue concentrations of dieldrin were  $0.05 \pm 0.14$  ppm for bottom feeders as compared to  $0.04 \pm 0.10$  ppm for predator species. Dieldrin was also detected in fish tissue at 60 percent of the 362 sites surveyed as part of the EPA National Survey of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median concentrations of dieldrin in fish tissues were 0.450, 0.028, and 0.004 ppm (wet weight), respectively. Mean dieldrin residues from the NSCRF study were highest in bottom feeders such as carp (0.045 ppm), white sucker (0.023 ppm), and channel catfish (0.015 ppm) as compared to predator species such as largemouth bass (0.005 ppm), smallmouth bass (0.002 ppm), and walleye (0.002 ppm) (Kuehl et al., 1994).

In 1993, three states (Arizona, Illinois, and Nebraska) had issued advisories for dieldrin contamination in fish (RTI, 1993). As of 1998, there were 23 advisories in effect in six states (Arizona, California, Colorado, Hawaii, Nebraska, and Texas) for this pesticide (U.S. EPA, 1999c). Dieldrin should be considered for inclusion in all state fish and shellfish contaminant monitoring programs in areas where its use as well as the use of aldrin have been extensive. States should contact their appropriate state agencies to obtain information on the historic uses of these two pesticides. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where dieldrin and aldrin were applied since dieldrin is a degradation product of aldrin. In suburban/urban watersheds, the degree of historic use of dieldrin and aldrin in domestic home and garden

applications should be evaluated. Sites in industrial watersheds should be reviewed to identify historic sites of dieldrin and aldrin production, formulation, or packaging facilities.

#### **4.3.2.5 Endosulfan—**

Endosulfan is a chlorinated cyclodiene pesticide that is currently in wide use primarily as a noncontact insecticide for seed and soil treatments (Appendix F). Two stereohomologues (I and II) exist and exhibit approximately equal effectiveness and toxicity (Worthing, 1991).

Endosulfan is highly toxic to laboratory animals and has been assigned to EPA Toxicity Class I (U.S. EPA, 1998d). To date, no studies have been found concerning carcinogenicity in humans after oral exposure to endosulfan (ATSDR, 1998c). EPA has classified endosulfan as Group E, evidence of noncarcinogenicity for humans (U.S. EPA, 1999b).

Agricultural runoff is the primary source of this pesticide in aquatic ecosystems. Endosulfan has been shown to be highly toxic to fish and marine invertebrates and is readily absorbed in sediments. It therefore represents a potential hazard in the aquatic environment (Sittig, 1980). However, data are insufficient to assess nationwide endosulfan contamination (NAS, 1991). Endosulfan has been included in one national fish contaminant monitoring program—the U.S. EPA 301(h) Program—the (U.S. EPA 301(h) Program—evaluated by the 1993 EPA Workgroup (Appendix E); however, no information was located related to its concentrations in fish or shellfish tissue.

In 1993, no consumption advisories were in effect for endosulfan I or II (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Endosulfan I and II should be considered for inclusion in all state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where endosulfan currently is used and was used historically. Sites in industrial watersheds should be reviewed to identify historic and current sites of endosulfan production, formulation, or packaging facilities.

#### **4.3.2.6 Endrin—**

Endrin is a chlorinated cyclodiene that historically was widely used as a broad spectrum pesticide. Endrin was first registered for use in the United States in 1951. However, recognition of its long-term persistence in soil and its high levels of mammalian toxicity led to restriction of its use beginning in 1964 and 1979 (U.S. EPA, 1980a; 44 FR 43632) and to final cancellation of its registration in 1984 (U.S. EPA, 1984a) (Appendix F).

Endrin is highly toxic to humans (EPA Toxicity Class I) (U.S. EPA, 1998d), with acute exposures affecting the central nervous system primarily (Sax, 1984). At present, evidence of both animal and human carcinogenicity of endrin is considered inadequate, and EPA has classified endrin in Group D, not classifiable as to human carcinogenicity insufficient information available (Appendix G) (IRIS, 1999).

Although endrin has been included in five national fish contaminant monitoring programs (Appendix E), it has not been found widely throughout the United States. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Endrin was detected in freshwater fish at only 29 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of endrin in 1984 were 0.22 and <0.01 ppm (wet weight), respectively (Schmitt et al. 1990). Endrin was also detected in freshwater and marine species at 11 percent of the 362 sites surveyed in the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median concentrations of endrin in fish tissues were 0.162 ppm, 0.002 ppm, and not detectable (wet weight), respectively. Mean endrin residues from the NSCRF study were highest in bottom feeders such as carp (0.0014 ppm), white sucker (0.0002 ppm), and channel catfish (0.009 ppm) as compared to predatory species such as largemouth bass (not detectable), smallmouth bass (not detectable), and walleye (not detectable) (Kuehl et al., 1994).

In 1993, no state had issued a fish advisory for endrin (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Endrin should be considered for inclusion in all state fish and shellfish contaminant monitoring programs in areas where its use has been extensive. States should contact their appropriate agencies to obtain information on the historic uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where endrin was used historically. Sites in industrial watersheds should be reviewed to identify historic sites of endrin production, formulation, or packaging facilities.

### 4.3.2.7 Heptachlor Epoxide—

Heptachlor epoxide is not a formulated pesticide but is a metabolic degradation product of the pesticides heptachlor and chlordane. It is also found as a contaminant in heptachlor and chlordane formulations (Appendix F). Heptachlor epoxide is also more toxic than either parent compound (ATSDR, 1993). Heptachlor has been used as a persistent, nonsystemic contact and ingested insecticide on soils (particularly for termite control) and seeds and as a household insecticide (Worthing, 1991). EPA suspended the major uses of heptachlor in 1978 (ATSDR, 1993). Acute exposures to high doses of heptachlor epoxide in humans can cause central nervous system effects (e.g., irritability, dizziness, muscle tremors, and convulsions (U.S. EPA, 1986c). In animals, liver, kidney, and blood disorders can occur (IRIS, 1999). Exposure to this compound

produced an increased incidence of liver carcinomas in rats and mice and hepatomas in female rats (IRIS, 1999). Heptachlor epoxide has been classified by EPA as a probable human carcinogen (B2) (Appendix G) (IRIS, 1999).

Heptachlor epoxide has been included in six national fish monitoring programs (Appendix E) and has been detected widely in freshwater finfish (Schmitt et al., 1990), but infrequently in bivalves and marine fish (NOAA, 1987, 1989a). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Heptachlor epoxide was detected in freshwater fish at 49 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of heptachlor epoxide in 1984 were 0.29 and 0.01 ppm (wet weight), respectively (Schmitt et al., 1990). Heptachlor epoxide also was detected in fish tissue at 16 percent of the 362 sites where it was surveyed in the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median concentrations of heptachlor epoxide were 0.063 ppm, 0.002 ppm, and not detectable (wet weight). It should be noted that one of the parent compounds, heptachlor was detected at only 2 percent of the 362 sites where it was surveyed at a maximum, arithmetic mean, and median concentration of 0.076, 0.0004 ppm, and not detectable, respectively. The five degradation products of chlordane were detected at from 27 to 77 percent of these same sites (see Section 4.3.2.1 for a discussion of chlordane). Mean heptachlor epoxide residues from the NSCRF study were highest in bottom feeders such as carp (0.004 ppm), white sucker (0.001 ppm), and channel catfish (0.0005 ppm) as compared to predator species such as largemouth bass (0.0003 ppm), smallmouth bass (0.00007 ppm), and walleye (0.0002 ppm) (Kuehl et al., 1994).

In 1993, only Nebraska had fish advisories for heptachlor epoxide contamination (RTI, 1993). As of 1998, there was only one advisory in effect, in Texas, for this pesticide degradation product (U.S. EPA, 1999c). Heptachlor epoxide should be considered for inclusion in all state fish and shellfish monitoring programs in areas where the use of heptachlor or chlordane have been extensive. States should contact their appropriate agencies to obtain information on the historic uses of these pesticides. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where heptachlor and chlordane were historically used since both of these pesticides degrade to heptachlor epoxide. In suburban/urban watersheds, the degree of historic use of heptachlor and chlordane in domestic home and garden applications should be evaluated. Sites in industrial watersheds also should be reviewed to identify historic sites of heptachlor and chlordane production, formulation, or packaging facilities.

### 4.3.2.8 Hexachlorobenzene—

Hexachlorobenzene is a fungicide that was widely used as a seed protectant in the United States until 1984 (Appendix F). The use of hexachlorobenzene and the presence of hexachlorobenzene residues in food are banned in many countries

including the United States (Worthing, 1991). Registration of hexachlorobenzene as a pesticide was voluntarily canceled in 1984 (Morris and Cabral, 1986).

The toxicity of this compound is minimal; it has been given an EPA toxicity classification of IV (i.e., oral LD<sub>50</sub> greater than 5,000 ppm in laboratory animals (U.S. EPA, 1998d). However, nursing infants are particularly susceptible to hexachlorobenzene poisoning as lactational transfer can increase infant tissue levels to two to five times maternal tissue levels (ATSDR, 1996). Hexachlorobenzene is a known animal carcinogen (ATSDR, 1996) and has been classified by EPA as a probable human carcinogen (B2) (Appendix G) (IRIS, 1999).

Of the chlorinated benzenes, hexachlorobenzene is the most widely monitored (Worthing, 1991). It was included as a target analyte in seven of the major monitoring programs reviewed by the 1993 Workgroup (Appendix E). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Hexachlorobenzene was detected in freshwater fish at 19 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of hexachlorobenzene in 1984 were 0.41 and <0.01 ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP on hexachlorobenzene in bottom-feeding and predator fish. The authors reported that there was no significant difference in residues in these two trophic groups. Mean tissue concentrations of HCB were 0.00 ± 0.01 and 0.01 ± 0.04 ppm, respectively, for bottom feeders and predator species. Hexachlorobenzene also was detected in fish tissue at 46 percent of the 362 sites where it was surveyed in the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median concentrations were 0.913 ppm, 0.006 ppm, and not detectable (wet weight), respectively. Mean hexachlorobenzene residues from the NSCRF study were highest in bottom feeders such as carp (0.0036 ppm), white sucker (0.0036 ppm), and channel catfish (0.0024 ppm) as compared to predator species such as largemouth bass (0.0002 ppm), smallmouth bass (0.0004 ppm), and walleye (0.0001 ppm) (Kuehl et al., 1994).

In 1993, Louisiana and Ohio had issued advisories for hexachlorobenzene contamination in fish and shellfish (RTI, 1993). As of 1988, there were three advisories in effect in these two states for this pesticide (U.S. EPA, 1999c). Hexachlorobenzene should be considered for inclusion in all state fish and shellfish monitoring programs. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where hexachlorobenzene was historically used. Sites in industrial watersheds also should be reviewed to identify historic sites of hexachlorobenzene as well as other organochlorine pesticide production, formulation, or packaging facilities since hexachlorobenzene was used as an intermediate in the chemical synthesis of many organochlorine pesticides.

#### 4.3.2.9 Lindane—

Lindane is a mixture of homologues of hexachlorocyclohexane ( $C_6H_6Cl_6$ ), whose major component ( $\geq 99$  percent) is the gamma isomer. It is commonly referred to as either  $\gamma$ -HCH (hexachlorocyclohexane) or  $\gamma$ -BHC (benzene hexachloride). Lindane is used primarily in seed treatments, soil treatments for tobacco transplants, foliage applications on fruit and nut trees and vegetables, and wood and timber protection. Lindane is used as a therapeutic scabicide, pediculicide, and ectoparasiticide for humans and animals (Merck Index 1989). Since 1985, many uses of lindane have been banned or restricted (see Appendix F) and its application is permitted only under supervision of a certified applicator (U.S. EPA, 1985c). In 1993, EPA issued a "Notice of Receipt of a Request for Amendments to Delete Uses" for several formulations of lindane provider, 99.5 percent technical, and dust concentrate, which would delete from the pesticide label most uses of lindane for agricultural crops and use on animals and humans (EPA 1993).

Lindane is a neurotoxin (assigned to EPA Toxicity Class II) (U.S. EPA, 1998d) and has been found to cause aplastic anemia in humans (Worthing, 1991). Lindane has been classified by EPA as a probable/possible human carcinogen (B2/C) (Appendix G) (U.S. EPA, 1999b).

Lindane has been included in seven major fish contaminant monitoring programs (Appendix E). This pesticide has been detected in freshwater fish (Schmitt et al., 1990) and in marine fish and bivalves (NOAA, 1987, 1989a) nationwide. In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Lindane was detected in freshwater fish at 47 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of lindane in 1984 were 0.40 and  $<0.01$  ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on lindane in bottom-feeding and predator fish. These authors reported there was no significant difference in residues in these two trophic groups of fish. Lindane also was detected in fish tissue at 42 percent of 362 sites surveyed in the EPA National Study of Chemical Residues in Fish (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median lindane concentrations were 0.083 ppm, 0.003 ppm, and not detectable (wet weight), respectively. Mean lindane residues from the NSCRF study were highest in bottom feeders such as carp (0.0043 ppm), white sucker (0.0017 ppm), and channel catfish (0.0032 ppm) as compared to predator species such as largemouth bass (0.00007 ppm), smallmouth bass (0.00015 ppm), and walleye (not detectable) (Kuehl et al., 1994).

In 1993, although it had been widely monitored and widely detected, no consumption advisories were in effect for lindane (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Lindane should be considered for inclusion in all state fish and shellfish monitoring programs in areas where its use has been extensive. States should contact their appropriate

agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where lindane was used historically. In suburban/urban watersheds, the degree of historic use of lindane in domestic home and garden applications should be evaluated. Sites in industrial watersheds should be reviewed to identify historic and current sites of lindane production, formulation, or packaging facilities.

#### 4.3.2.10 Mirex—

Mirex is a chlorinated cyclodiene pesticide that was used in large quantities in the United States from 1962 through 1975 primarily for control of fire ants in the Southeast and Gulf Coast states and, more widely, under the name Dechlorane as a fire retardant and polymerizing agent in plastics (Kaiser, 1978; Kutz et al., 1985) (Appendix F).

Mirex has been assigned to EPA Toxicity Class II on the basis of an oral LD<sub>50</sub> in rats of 368 mg/kg (ATSDR, 1995; U.S. EPA, 1998d) (Appendix F). Mirex has been assigned a carcinogenicity classification of group B2, probable human carcinogen (HEAST, 1997). EPA instituted restrictions on the use of mirex in 1975, and, thereafter, the U.S. Department of Agriculture (USDA) suspended the fire ant control program (Hodges, 1977).

Mirex has been included in seven major fish contaminant monitoring programs (Appendix E). It has been found primarily in the Southeast, Gulf Coast, and the Great Lakes regions (Kutz et al., 1985; NAS, 1991; Schmitt et al., 1990). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (NCBP) (Schmitt et al., 1990). Mirex was detected in freshwater fish at 13 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of mirex in 1984 were 0.44 and <0.01 ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on mirex in bottom-feeding and predator fish. These authors reported there was no significant difference in residues in these two trophic groups of fish. Mean tissue concentrations of mirex were  $0.00 \pm 0.04$  and  $0.01 \pm 0.05$  ppm, respectively, for bottom feeders and predator species. Mirex also was detected in fish tissue at 38 percent of 362 sites surveyed in the EPA National Study of Chemical Residues in Fish (NSCRF) (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median mirex concentrations were 0.225 ppm, 0.004 ppm, and not detectable (wet weight), respectively. Mean mirex residues from the EPA NSCRF study were highest in bottom feeders such as carp (0.0037 ppm), white sucker (0.0044 ppm), and channel catfish (0.0146 ppm) as compared to predator species such as largemouth bass (0.0002 ppm), smallmouth bass (0.002 ppm), and walleye (0.00008 ppm) (Kuehl et al., 1994).

In 1993, three states (New York, Ohio, and Pennsylvania) had issued fish advisories for mirex (RTI, 1993). As of 1998, there were 11 advisories in effect

in these same three states for this pesticide (U.S. EPA, 1999c). New York has a statewide advisory in effect for mergansers. Mirex should be considered for inclusion in all state fish and shellfish monitoring programs in areas where its use has been extensive. States should contact their appropriate agencies to obtain information on the historic uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where mirex was used historically. In suburban/urban watersheds, the degree of historic use of mirex in domestic home and garden applications should be evaluated. Sites in industrial watersheds should be reviewed to identify historic sites of mirex production, formulation, or packaging facilities.

#### **4.3.2.11 Toxaphene—**

Toxaphene is an organochlorine pesticide composed of a complex mixture of chlorinated camphenes (chlorinated bornanes and some bornenes) that was first registered for use in the United States in 1947. It was commercially produced by the chlorination of camphenes derived from pine trees. It has been estimated that the commercial mixture of toxaphene contained at least 670 congeners with the majority of these having 6 to 10 chlorines (Jansson and Wideqvist, 1983). Historically, this compound was used in the United States as an insecticide primarily on cotton (Hodges, 1977). In addition, toxaphene was used as a piscicide for rough fish in the 1950s and 1960s in North America and was the replacement for DDT after DDT's use was severely restricted in 1972 (Saleh, 1991). Partly as a consequence of the ban on the use of DDT imposed in 1972, toxaphene was for many years the most heavily used pesticide in the United States (Eichers et al., 1978). In 1982, toxaphene's registration for most uses was canceled (47 FR 53784) and all uses were banned in 1990 (55 FR 31164-31174). Toxaphene is a global pollutant whose chemical-physical properties make it a candidate for long-range atmospheric transport via the cold condensation effect once it is released into the environment (Wania and Mackay, 1993, 1996).

Like many of the other organochlorine pesticides, toxaphene has been assigned to EPA Toxicity Class II (U.S. EPA, 1998d) (Appendix F). Some components of toxaphene may accumulate in body fat. Toxaphene has been classified by EPA as a probable human carcinogen (B2) (Appendix G) (IRIS, 1999).

Toxaphene has been included in four major fish contaminant monitoring programs (Appendix E). It has been detected frequently in both freshwater fish (Schmitt et al., 1990) and estuarine species (NOAA, 1989a) but is only consistently found in Georgia, Texas, and California (NAS, 1991). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 321 composite samples of whole fish from 112 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt et al., 1990). Toxaphene was detected in freshwater fish at 69 percent of 112 stations sampled in the NCBP study. Maximum and geometric mean tissue concentrations of toxaphene in 1984 were 8.2 and 0.14 ppm (wet weight), respectively (Schmitt et al., 1990). Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on toxaphene in bottom-feeding and predatory fish species. These authors reported there was no significant difference

in residues in these two trophic groups of fish. Mean tissue concentrations of toxaphene were  $0.19 \pm 0.63$  and  $0.17 \pm 0.35$  ppm, respectively, for bottom feeders and predator species.

In 1993, two states (Arizona and Texas) had fish advisories in effect for toxaphene (RTI, 1993). As of 1988, there were six advisories in effect in four states (Arizona, Georgia, Oklahoma, and Texas) for this pesticide (U.S. EPA, 1999c). Toxaphene should be considered for inclusion in all state fish and shellfish monitoring programs in areas where its use has been extensive. States should contact their appropriate agencies to obtain information on the historic uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where toxaphene was used historically. Sites in industrial watersheds should be reviewed to identify historic sites of toxaphene production, formulation, or packaging facilities.

### 4.3.3 Organophosphate Pesticides

The following organophosphate pesticides are recommended as target analytes in screening studies: chlorpyrifos, diazinon, disulfoton, ethion, and terbufos (Appendix E). These pesticides share two distinct features that differentiate them from the organochlorines. Organophosphate pesticides are generally more acutely toxic to vertebrates than organochlorine pesticides and exert their toxic action by inhibiting the activity of cholinesterase (ChE), one of the vital nervous system enzymes. In addition, organophosphates are chemically unstable (they are all slowly hydrolyzed by water) and thus are less persistent in the environment. It is this latter feature that made them attractive alternatives to the organochlorine pesticides that were used extensively in agriculture from the 1940s to the early 1970s.

With the exception of chlorpyrifos, none of the organophosphates has been included in any of the national fish contaminant monitoring programs evaluated by the EPA 1993 Workgroup and none of these pesticides (including chlorpyrifos) has triggered state fish consumption advisories. All of the organophosphate pesticides have active pesticide registrations and have been recommended for monitoring because they have an EPA Toxicity Classification of I or II (Appendix F), BCFs >300, and a half-life of 30 days or more in the environment and their use profiles suggest they could be potential problems in some agricultural watersheds.

The target organophosphates are used in agriculture throughout the United States, particularly in areas under intensive cultivation (row crops, orchards, fruits, and vegetables). Bioconcentration studies indicate they can accumulate in fish and, because they are known human neurotoxins, the potential exists for human health effects from consuming contaminated fish. For this reason, federal regulations are in effect that set maximum application rates and minimize use near waterbodies. At the time of this writing, no fish consumption advisories for these target analytes have yet been issued; however, state agencies should be aware of special circumstances that could result in their accumulation in fish. In

addition to chemical spills and misapplications, heavy and repeated rainfall shortly after application may wash pesticides off of plants and into streams. Signs of acute organophosphate pollution may include erratic swimming behavior in fish or fish kills.

States should contact their appropriate agencies to obtain information on both the historic and current uses of these pesticides. With the exception of ethion, which is used almost exclusively on citrus, the target organophosphates are used on a wide variety of crops. In addition, chlorpyrifos and diazinon have significant uses in domestic and commercial pest control in suburban/urban areas (Robinson et al., 1994). If a state determines that high concentrations of these pesticides may be present in its agricultural watersheds, sampling should be conducted during late spring or early summer within 1 to 2 months following pesticide application to maximize detection of these compounds in fish tissues. In general, the organophosphates are degraded relatively rapidly in the environment and metabolized relatively rapidly by fish, so timing of the sampling program is a more important consideration for this class of pesticides. Additional discussion of appropriate sampling times for fish contaminant monitoring programs is provided in Section 6.1.1.5.

All of the target organophosphates are members of the organothiophosphate group of insecticides. They are all metabolized in the liver to their active form, referred to as an "oxon" (e.g., chlorpyrifos is activated to chlorpyrifos oxon) (Klaasen, 1996). The oxons are approximately 300- to 1,000-fold more toxic than the parent compounds; however, they are also less lipid-soluble than the parent compounds and, therefore, are expected to be less likely to bioaccumulate in fish tissue. In another laboratory study where chlorpyrifos was fed to channel catfish, only chlorpyrifos and its inactive metabolites were found; the oxon was not detected in any tissue (Barron et al., 1991). No information is available on the presence of the oxon metabolites in fish tissue for the other organophosphates.

**Note:** The potential human toxicity of the organophosphates is undergoing reassessment by EPA at this time as a result of the provisions of the Food Quality Protection Act of 1996. For more information, consult the EPA Office of Pesticide Programs webpage available on the Internet at:

<http://www.epa.gov.pesticides/op>.

### 4.3.3.1 Chlorpyrifos—

This organophosphate pesticide was first introduced in 1965 to replace the more persistent organochlorine pesticides (e.g., DDT) (U.S. EPA, 1986c) and has been used for a broad range of insecticide applications (Appendix F). Chlorpyrifos is used primarily to control soil and foliar insects on cotton, peanuts, and sorghum (Worthing, 1991; U.S. EPA, 1986c). Chlorpyrifos is also used to control root-infesting and boring insects on a variety of fruits (e.g., apples, bananas, citrus, grapes), nuts (e.g., almonds, walnuts), vegetables (e.g., beans, broccoli, brussel sprouts, cabbage, cauliflower, peas, and soybeans), and field crops (e.g., alfalfa and corn) (U.S. EPA, 1984c). As a household insecticide, chlorpyrifos has been

used to control ants, cockroaches, fleas, and mosquitoes (Worthing, 1991) and is registered for use in controlling subsurface termites in California (U.S. EPA, 1983a). Based on use application, 48 percent of chlorpyrifos use is agricultural and 52 percent is nonagricultural (U.S. EPA, 2000b). Chlorpyrifos is also used by the general public for home, lawn, and garden insect control (ATSDR, 1997).

Note: As a result of the reassessment conducted under the Food Quality Act of 1996, use patterns of chlorpyrifos will change significantly by the end of 2001. In particular, virtually all indoor and outdoor residential use will end, as well as all agricultural use on tomatoes. Agricultural use of chlorpyrifos on apples and grapes will be reduced substantially (U.S. EPA, 2000b).

Chlorpyrifos has a moderate mammalian toxicity and has been assigned to EPA Toxicity Class II based on oral feeding studies (U.S. EPA, 1998d). No carcinogenicity was found in chronic feeding studies with rats, mice, and dogs (U.S. EPA, 1983a). Because chlorpyrifos did not increase the incidence of cancer in feeding studies on rats and mice (U.S. EPA, 1999b, U.S. EPA, 2000b) EPA has classified chlorpyrifos in Group E (Appendix G) (U.S. EPA, 2000b). Experimental evidence indicates this compound bioaccumulates in rainbow trout (BCF from 1,280 to 3,903) (U.S. EPA, 1993a).

Chlorpyrifos has been included in one national monitoring program reviewed by the EPA 1993 Workgroup, the EPA National Study of Chemical Residues in Fish (NSCRF) (see Appendix E). In this study, chlorpyrifos was detected at 26 percent of sites sampled nationally (U.S. EPA, 1992c, 1992d). Eighteen percent of the sites with relatively high concentrations (0.0025 to 0.344 ppm) were scattered throughout the East, Midwest, and in California; the highest mean concentrations detected (0.060 to 0.344 ppm) were found either in agricultural areas or in urban areas with a variety of nearby industrial sources. Maximum, arithmetic mean, and median tissue concentrations (wet weight) of chlorpyrifos were 0.344 ppm, 0.004 ppm, and not detectable, respectively. Mean chlorpyrifos residues from the NSCRF study were highest in bottom feeders such as carp (0.0082 ppm), white sucker (0.0018 ppm), and channel catfish (0.007 ppm) as compared to predator species such as largemouth bass (0.00028 ppm), smallmouth bass (0.00008 ppm), and walleye (0.00004 ppm) (Kuehl et al., 1994). It should be noted that this national study did not specifically target agricultural sites where this pesticide historically had been used or is currently used. Chlorpyrifos residues in fish could be much higher if sampling were targeted for pesticide runoff, especially during the period immediately after field application.

In 1993, no consumption advisories were in effect for chlorpyrifos (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Chlorpyrifos should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where chlorpyrifos is currently used or was used historically. In suburban/urban water-

sheds, the degree of historic and current use of chlorpyrifos in domestic home and garden applications should be evaluated. Sites in industrial watersheds also should be reviewed to identify historic and current sites of chlorpyrifos production, formulation, or packaging facilities.

#### **4.3.3.2 Diazinon—**

Diazinon is a phosphorothiate insecticide and nematicide that was first registered in 1952 for control of soil insects and pests of fruits, vegetables, tobacco, forage, field crops, range, pasture, grasslands, and ornamentals; for control of cockroaches and other household insects; for control of grubs and nematodes in turf; as a seed treatment; and for fly control (U.S. EPA, 1986d). Diazinon is also used by the general public for home, lawn, and garden insect control (Appendix F) (ATSDR, 1996).

Diazinon is moderately toxic to mammals and has been assigned to EPA Toxicity Class II based on oral toxicity tests (U.S. EPA, 1998d) (Appendix F). Diazinon was not found to be carcinogenic in rats and mice (ATSDR, 1996). Because of inadequate evidence of carcinogenicity, EPA has classified diazinon as “not likely to be a human carcinogen” (Appendix G) (U.S. EPA, 1998d). This compound is also highly toxic to birds, fish, and other aquatic invertebrates (U.S. EPA, 1986d).

Diazinon was not included in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). Experimental evidence indicates this compound accumulates in trout (BCF of 542) (U.S. EPA, 1993a).

In 1993, no consumption advisories were in effect for diazinon (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Diazinon should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where diazinon is currently used or was used historically. In suburban/urban watersheds, the degree of historic and current use of diazinon in domestic home and garden applications should be evaluated. Sites in industrial watersheds should be reviewed to identify historic and current sites of diazinon production, formulation, or packaging facilities.

#### **4.3.3.3 Disulfoton—**

Disulfoton is a multipurpose systemic insecticide and acaricide first registered in 1958 for use as a side dressing, broadcast, or foliar spray in the seed furrow to control many insect and mite species and as a seed treatment for sucking insects (Appendix F) (*Farm Chemicals Handbook*, 1989).

Disulfoton is highly toxic to all mammalian systems and has been assigned to EPA Toxicity Class I on the basis of all routes of exposure (U.S. EPA, 1998d).

Disulfoton was not found to be carcinogenic in dogs, rats, or mice (ATSDR, 1995). Because of inadequate evidence of carcinogenicity, EPA has classified disulfoton as Group E, evidence of noncarcinogenicity for humans (Appendix G) (U.S. EPA, 1999b).

Disulfoton was not included in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). Experimental evidence indicates this compound accumulates in fish (BCF from 460 to 700) (U.S. EPA, 1993a).

In 1993, no consumption advisories were in effect for disulfoton (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Disulfoton should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where disulfoton currently is used or was used historically. Sites in industrial watersheds also should be reviewed to identify historic and current sites of disulfoton production, formulation, or packaging facilities.

### 4.3.3.4 Ethion—

Ethion is a multipurpose insecticide and acaricide that has been registered since 1965 for use on a wide variety of nonfood crops (turf, evergreen plantings, and ornamentals), food crops (seed, fruit, nut, fiber, grain, forage, and vegetables), and for domestic outdoor uses around dwellings and for lawns (Appendix F) (*Farm Chemicals Handbook*, 1989). Application to citrus crops accounts for 86 to 89 percent of the ethion used in the United States. The remaining 11 to 14 percent is applied to cotton and a variety of fruit and nut trees and vegetables. Approximately 55 to 70 percent of all domestically produced citrus fruits are treated with ethion (U.S. EPA, 1989e).

Acute oral toxicity studies have shown that technical-grade ethion is moderately toxic to mammals (EPA Toxicity Class II) (U.S. EPA, 1998d). Ethion was not found to be carcinogenic in rats and mice (U.S. EPA, 1989e). EPA has classified ethion in Group E—evidence of noncarcinogenicity for humans (Appendix G) (U.S. EPA, 1999b).

Ethion was not included in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). Experimental evidence indicates this compound accumulates in bluegill sunfish (BCF from 880 to 2,400) (U.S. EPA, 1993a).

In 1993, no consumption advisories were in effect for ethion (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Ethion should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States

should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where ethion currently is used or was used historically. In suburban/urban watersheds, the degree of historic and current use of ethion in domestic home and garden applications should be evaluated. Sites in industrial watersheds also should be reviewed to identify historic and current sites of ethion production, formulation, or packaging facilities.

### 4.3.3.5 Terbufos—

Terbufos is a systemic organophosphate insecticide and nematicide registered in 1974 principally for use on corn, sugar beets, and grain sorghum. The primary method of application involves direct soil incorporation of a granular formulation (*Farm Chemicals Handbook*, 1989). Two soil metabolites of terbufos, terbufos sulfoxide and terbufos sulfone, are also toxic to humans and are found at sites where terbufos has been applied (U.S. EPA, 1995)

Terbufos is highly toxic to humans and has been assigned to EPA Toxicity Class I (U.S. EPA, 1998d) (Appendix F). Terbufos was not found to be carcinogenic in rats and mice (U.S. EPA, 1995j). EPA has assigned terbufos to carcinogenicity classification E, evidence of noncarcinogenicity for humans (U.S. EPA, 1998d) (Appendix G). Terbufos is also highly toxic to birds, fish, and other aquatic invertebrates (U.S. EPA, 1985d).

Terbufos was not included in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). Experimental evidence indicates this compound accumulates in fish (BCF from 320 to 1,400) (U.S. EPA, 1993a).

In 1993, no consumption advisories were in effect for terbufos (RTI, 1993). As of 1998, there were no advisories in effect for this pesticide (U.S. EPA, 1999c). Terbufos and its toxic metabolites should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where terbufos currently is used or was used historically. Sites in industrial watersheds also should be reviewed to identify historic and current sites of terbufos production, formulation, or packaging facilities.

### 4.3.4 Chlorophenoxy Herbicides

Chlorophenoxy herbicides, which include oxyfluorfen, are nonselective foliar herbicides that are most effective in hot weather (Ware, 1978).

#### 4.3.4.1 Oxyfluorfen—

Oxyfluorfen is a pre- and postemergence herbicide with an active registration that has been registered since 1979 for use to control a wide spectrum of annual broadleaf weeds and grasses in apples, artichokes, corn, cotton, jojoba, tree fruits, grapes, nuts, soybeans, spearmint, peppermint, and certain tropical plantation and ornamental crops (Appendix F) (*Farm Chemicals Handbook*, 1989).

Oxyfluorfen is of low toxicity to mammals (oral LD<sub>50</sub> in rats >5,000 mg/kg) and has been assigned to EPA Toxicity Class IV (U.S. EPA, 1998d) (Hayes and Lawes, 1991). There is also evidence of carcinogenicity (liver tumors) in mice (U.S. EPA, 1993a) and therefore oxyfluorfen has been classified by EPA as a possible human carcinogen (C) (Appendix G) (U.S. EPA, 1999b).

Oxyfluorfen was not included in any national fish contaminant monitoring program evaluated by the EPA 1993 Workgroup (Appendix E). Experimental evidence indicates this herbicide accumulates in bluegill sunfish (BCF from 640 to 1,800) (U.S. EPA, 1993a).

In 1993, no consumption advisories were in effect for oxyfluorfen (RTI, 1993). As of 1998, there were no advisories in effect for this herbicide (U.S. EPA, 1999c). Oxyfluorfen should be considered for inclusion in state fish and shellfish contaminant monitoring programs in areas where its use is or has been extensive. States should contact their appropriate agencies to obtain information on the historic and current uses of this pesticide. Monitoring sites in agricultural watersheds should be reviewed to determine the application rate and acreage where oxyfluorfen currently is used or was used historically. Sites in industrial watersheds also should be reviewed to identify historic and current sites of oxyfluorfen production, formulation, or packaging facilities.

#### 4.3.5 Polycyclic Aromatic Hydrocarbons

PAHs are base/neutral organic compounds that have a fused ring structure of two or more benzene rings. PAHs are also commonly referred to as polynuclear aromatic hydrocarbons (PNAs). PAHs with two to five benzene rings (i.e., 10 to 24 skeletal carbons) are generally of greatest concern for environmental and human health effects (Benkert, 1992). These PAHs have been identified as the most important with regard to human exposure (ATSDR, 1995):

- Acenaphthene
- Acenaphthylene
- Anthracene
- Benz[*a*]anthracene
- Benzo[*a*]pyrene
- Benzo[*e*]pyrene
- Benzo[*b*]fluoranthene
- Benzo[*k*]fluoranthene
- Benzo[*j*]fluoranthene
- Benzo[*g,h,i*]perylene
- Chrysene
- Dibenz[*a,h*]anthracene
- Fluoranthene
- Fluorene
- Indeno[*1,2,3-cd*]pyrene
- Phenanthrene

- Pyrene.

The metabolites of many of the high-molecular-weight PAHs (e.g., benz[*a*]anthracene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz[*a,h*]anthracene, indeno[1,2,3-*cd*]pyrene, and benzo[*g,h,i*]perylene) have been shown in laboratory test systems to be carcinogens, cocarcinogens, teratogens, and/or mutagens (Moore and Ramamoorthy, 1984; ATSDR 1995). Benzo[*a*]pyrene, one of the most widely occurring and potent PAHs, and six other PAHs (e.g., benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, dibenz[*a,h*]anthracene, indeno[1,2,3-*cd*]pyrene) have been classified by EPA as probable human carcinogens (B2) (IRIS, 1999). Evidence for the carcinogenicity of PAHs in humans comes primarily from epidemiologic studies that have shown an increased mortality due to lung cancer in humans exposed to PAH-containing coke oven emissions, roof-tar emissions, and cigarette smoke (ATSDR, 1995).

PAHs are ubiquitous in the environment and usually occur as complex mixtures with other toxic chemicals. They are components of crude and refined petroleum products and of coal. They are also produced by the incomplete combustion of organic materials. Many domestic and industrial activities involve pyrosynthesis of PAHs, which may be released into the environment in airborne particulates or in solid (ash) or liquid byproducts of the pyrolytic process. Domestic activities that produce PAHs include cigarette smoking, home heating with wood or fossil fuels, waste incineration, broiling and smoking foods, and use of internal combustion engines. Industrial activities that produce PAHs include wood preserving, coal coking; production of carbon blacks, creosote, and coal tar; petroleum refining; synfuel production from coal; and use of Soderberg electrodes in aluminum smelters and ferrosilicum and iron works (ATSDR, 1995; Neff, 1985). Historic coal gasification sites have also been identified as significant sources of PAH contamination (ATSDR, 1995).

Major sources of PAHs found in marine and fresh waters include biosynthesis (restricted to anoxic sediments), spillage and seepage of fossil fuels, discharge of domestic and industrial wastes, atmospheric deposition, and runoff (Neff, 1985). Urban stormwater runoff contains PAHs from leaching of asphalt roads, wearing of tires, deposition from automobile exhaust, and oiling of roadsides and unpaved roadways with crankcase oil (ATSDR, 1995; MacKenzie and Hunter, 1979). Solid PAH-containing residues from activated sludge treatment facilities have been disposed of in landfills or in the ocean (ocean dumping was banned in 1989). Although liquid domestic sewage contains <1 µg/L total PAH, the total PAH content of industrial sewage is 5 to 15 µg/L (Borneff and Kunte, 1965) and that of sewage sludge is 1 to 30 mg/kg (Grimmer et al., 1978; Nicholls et al., 1979).

In most cases, there is a direct relationship between PAH concentrations in river water and the degree of industrialization and human activity in the surrounding watersheds. Rivers flowing through heavily industrialized areas may contain 1 to

5 ppb total PAH, compared to unpolluted river water, ground water, or seawater that usually contains less than 0.1 ppb PAH (Neff, 1979).

PAHs can accumulate in aquatic organisms from water, sediments, and food. BCFs of PAHs in fish, crustaceans, and bivalves have frequently been reported to be in the range of 12 to 9,200 for fish, 200 to 134,248 for crustaceans, and 8 to 242 for bivalves based on short-term exposure studies typically less than 7 days duration (Eisler, 1987). In general, bioconcentration was greater for the higher molecular weight PAHs than for the lower molecular weight PAHs. Biotransformation by the mixed function oxidase system in the fish liver can result in the formation of carcinogenic and mutagenic intermediates, and exposure to PAHs has been linked to the development of tumors in fish (Eisler, 1987). The ability of fish to metabolize PAHs probably explains why benzo[a]pyrene frequently is not detected or is found only at very low concentrations in fish from areas heavily contaminated with PAHs (Varanasi and Gmur, 1980, 1981).

Sediment-associated PAHs can be accumulated by bottom-dwelling invertebrates and fish (Eisler, 1987). For example, Great Lakes sediments containing elevated levels of PAHs were reported by Eadie et al. (1983) to be the source of the body burdens of the compounds in bottom-dwelling invertebrates. Similarly, Varanasi et al. (1985) found that benzo[a]pyrene was accumulated in fish, amphipod crustaceans, shrimp, and clams when estuarine sediment was the source of the compound. Approximate tissue-to-sediment ratios were 0.6 to 1.2 for amphipods, 0.1 for clams, and 0.05 for fish and shrimp. Although fish and most crustaceans evaluated to date have the mixed function oxidase system required for biotransformation of PAHs, many molluscs lack this system and are unable to metabolize PAHs efficiently (Varanasi et al., 1985). More important, PAHs induce mixed function oxidase enzymes (and thus their own biotransformation) in fish and other vertebrates, but not in molluscs and crustaceans (Stegeman and Lech, 1991). The resulting dramatic difference in biotransformation means that in PAH-contaminated waters, fish may show little or no accumulation of PAHs, while bivalve molluscs and crustaceans are heavily contaminated. Varanasi et al. (1985) ranked benzo[a]pyrene metabolism by aquatic organisms as follows: fish > shrimp > amphipod crustaceans > clams. Half-lives for elimination of PAHs in fish ranged from less than 2 days to 9 days (Niimi, 1987). NAS (1991) reported that PAH contamination in bivalves has been found in all areas of the United States. If PAHs are selected as a target analyte to be monitored at a site, primary preference should be given to selection of a bivalve mollusc (clam, oyster, mussel) as the target species, secondary preference should be given to a crustacean (shrimp, lobster, crab) (if available), and finfish should be given the lowest priority for selection as the target species. This ranking of the preferred target species for PAH analysis assumes that a bivalve mollusc and crustacean are available at the sampling site and that these species are eaten by the consumer population of concern.

In 1993, three states (Massachusetts, Michigan, and Ohio) had issued advisories for PAH contamination in finfish (RTI, 1993). As of 1998, there were five advisories in effect in four states (Massachusetts, Michigan, Ohio, and

Washington) for PAHs (U.S. EPA, 1999c). Monitoring sites in industrial and suburban/urban watersheds should be reviewed to identify current and historic sites of waste incinerators, coal gasification facilities, petroleum refineries, and creosote, coal tar, coal coking, and wood preservative facilities that are potential sources for PAH releases to the environment. Sites of petroleum spills should also be reviewed.

The EPA and others have developed a relative potency estimate approach for the PAHs (Nisbet and LaGoy, 1992; U.S. EPA, 1993c). Using this approach, the cancer potency of 14 carcinogenic PAHs can be estimated based on their relative potency to benzo[*a*]pyrene. Toxicity equivalence factors (TEF) for benzo[*a*]pyrene and the other 14 PAHs based on carcinogenicity are discussed in Section 5.3.2.4.

Although several PAHs have been classified as probable human carcinogens (Group B2), benzo[*a*]pyrene is the only PAH for which an oral CSF is currently available in IRIS (1999). It is recommended that, in both screening and intensive studies, tissue samples be analyzed for benzo[*a*]pyrene and the other 14 PAHs for which TEFs are available and that the relative potencies given for these PAHs (Nisbet and LaGoy, 1992; U.S. EPA, 1993c) be used to calculate a potency equivalency concentration for each sample for comparison with the recommended SVs for benzo[*a*]pyrene (see Section 5.3.2.4).

### 4.3.6 Polychlorinated Biphenyls (Total)

PCBs are base/neutral compounds that are formed by the direct chlorination of biphenyl. PCBs are closely related to many chlorinated hydrocarbon pesticides (e.g., DDT, dieldrin, and aldrin) in their chemical, physical, and toxicologic properties and in their widespread occurrence in the aquatic environment (Nimmo, 1985). There are 209 different PCB compounds, termed congeners, based on the possible chlorine substitution patterns. In the United States, mixtures of various PCB congeners were formulated for commercial use under the trade name Aroclor on the basis of their percent chlorine content. For example, a common PCB mixture, Aroclor 1254, has an average chlorine content of 54 percent by weight (Nimmo, 1985).

Unlike the organochlorine pesticides, PCBs were never intended to be released directly into the environment; most uses were in closed industrial systems. Important properties of PCBs for industrial applications include thermal stability, fire and oxidation resistance, and solubility in organic compounds (Hodges, 1977). PCBs were used as insulating fluids in electrical transformers and capacitors, as plasticizers, as lubricants, as fluids in vacuum pumps and compressors, and as heat transfer and hydraulic fluids (Hodges, 1977; Nimmo, 1985). Although use of PCBs as a dielectric fluid in transformers and capacitors was generally considered a closed-system application, the uses of PCBs, especially during the 1960s, were broadly expanded to many open systems where losses to the environment were likely. Heat transfer systems, hydraulic fluids in die cast machines, and uses in specialty inks are examples of more open-ended

applications that resulted in serious contamination in fish near industrial discharge points (Hesse, 1976).

Although PCBs were once used extensively by industry, their production and use in the United States were banned by the EPA in July 1979 (Miller, 1979). Prior to 1979, the disposal of PCBs and PCB-containing equipment was not subject to federal regulation. Prior to regulation, of the approximately 1.25 billion pounds purchased by U.S. industry, 750 million pounds (60 percent) were still in use in capacitors and transformers, 55 million pounds (4 percent) had been destroyed by incineration or degraded in the environment, and over 450 million pounds (36 percent) were either in landfills or dumps or were available to biota via air, water, soil, and sediments (Durfee et al., 1976).

PCBs are extremely persistent in the environment and are bioaccumulated throughout the food chain (Eisler, 1986; Worthing, 1991). There is evidence that PCB health risks increase with increased chlorination because more highly chlorinated PCBs are retained more efficiently in fatty tissues (IRIS, 1999). However, individual PCB congeners have widely varying potencies for producing a variety of adverse biological effects including hepatotoxicity, cardiovascular toxicity, developmental toxicity, immunotoxicity, neurotoxicity, and carcinogenicity. The non-ortho-substituted coplanar PCB congeners, and some of the mono-ortho-substituted congeners, have been shown to exhibit "dioxin-like" effects (Golub et al., 1991; Kimbrough and Jensen, 1989; McConnell, 1980; Poland and Knutson, 1982; Safe, 1985, 1990; Tilson et al., 1990; U.S. EPA 1993c; Van den Berg et al., 1998). The neurotoxic effects of PCBs appear to be associated with some degree of ortho-chlorine substitution. There is increasing evidence that many of the toxic effects of PCBs result from alterations in hormonal function. Because PCBs can act directly as hormonal agonists or antagonists, PCB mixtures may have complex interactive effects in biological systems (Korach et al., 1988; Safe et al., 1991; Shain et al., 1991; U.S. EPA, 1993c). Because of the lack of sufficient toxicologic data, EPA has not developed quantitative estimates of health risk for specific congeners; however, 12 dioxin-like congeners have been assigned TEFs and may be evaluated as contributing to dioxin health risk (Van den Berg et al., 1998). PCB mixtures have been classified as probable human carcinogens (Group B2) (Appendix G) (IRIS, 1999; U.S. EPA, 1988a).

PCB mixtures have been shown to cause adverse developmental effects in experimental animals (ATSDR, 1998b). Data are inconclusive in regard to developmental effects in humans. Several studies in humans have suggested that PCB exposure may cause adverse developmental effects in children and in developing fetuses (ATSDR, 1998b). These include lower IQ scores (Jacobson and Jacobson, 1996), low birth weight (Rylander et al., 1998), and lower behavior assessment scores (Lonky et al., 1996). However, study limitations, including lack of control for confounding variables, deficiencies in the general areas of exposure assessment, selection of exposed and control subjects, and the comparability of exposed and control samples obscured interpretation of these results (ATSDR, 1998b).

PCBs, total or as Aroclors, have been included in seven major fish contaminant monitoring programs evaluated by the 1993 EPA Workgroup (Appendix E). A summary of the U.S. Fish and Wildlife Service National Contaminants Biomonitoring Program (NCBP) data from 1976 through 1984 indicated a significant downward trend in the geometric mean concentration (wet weight basis) of total PCBs (from 0.89 ppm in 1976 to 0.39 ppm in 1984); however, PCB residues in fish tissue remain widespread, being detected at 91 percent of the sites monitored in 1984 (Schmitt et al., 1990). Maximum total PCB tissue residue concentrations during this same period also declined, from 70.6 ppm in 1976 to 6.7 ppm in 1984. Coinciding declines in tissue residue concentrations of three Aroclors (1248, 1254, and 1260) were also observed. Kidwell et al. (1995) conducted an analysis of all 1984-1985 data from the NCBP study on the three Aroclors in bottom-feeding and predatory fish species. These authors reported there was no significant difference in residues in these two trophic groups of fish for Aroclor 1248 and 1254; however, there were significantly higher concentrations of Aroclor 1260 in predator species as compared to bottom feeders. Mean tissue concentrations of Aroclor 1248, 1254, and 1260 were  $0.06 \pm 0.32$ ,  $0.21 \pm 0.39$ , and  $0.14 \pm 0.24$  ppm, respectively, for bottom feeders (e.g., carp, white suckers, and channel catfish) and  $0.08 \pm 0.31$ ,  $0.35 \pm 0.69$ , and  $0.23 \pm 0.38$  ppm, respectively, for predator species (e.g., rainbow, brown, brook, and lake trout, largemouth bass, and walleye).

Total PCBs also were detected at 91 percent of 374 sites surveyed in the EPA National Study of Chemical Residues in Fish (NSCRF) (U.S. EPA, 1992c, 1992d). Maximum, arithmetic mean, and median total PCB concentrations (wet weight) reported were 124, 1.89, and 0.209 ppm, respectively. As is shown in Table 4-6, the tri-, tetra-, penta-, hexa-, and heptachlorobiphenyls were detected in fish tissue samples at >50 percent of the NSCRF sites. Mean tissue concentrations were highest for the tetra- and pentachlorobiphenyls with concentrations of 0.696, 0.565, and 0.356 ppm, respectively. The median fish tissue concentrations were highest for the hexa- followed by the pentachlorobiphenyls with concentrations of 0.077 and 0.072 ppm, respectively.

With respect to sources of these compounds, PCBs were detected in all parts of the country with the highest concentrations being associated with paper mills, refinery/other industry sites, Superfund sites, wood preserving facilities, and industrial/urban areas. Mean total PCB concentrations from the NSCRF study were highest in bottom feeders (whole fish) such as carp (2.94 ppm), white sucker (1.7 ppm), and channel catfish (1.3 ppm) as compared to predator species (fillet samples) such as largemouth bass (0.23 ppm), smallmouth bass (0.5 ppm), and walleye (0.37 ppm) (Kuehl et al., 1994).

In 1993, PCB contamination in fish and shellfish resulted in the issuance of 328 advisories in 31 states and the U.S. territory of American Samoa (Figure 4-3) (RTI, 1993). As of 1998, there were 679 advisories in effect in 36 states and the U.S. territory of American Samoa for this compound (Figure 4-3) (U.S. EPA, 1999c.). In addition, two states (Indiana and New York) and the District of Columbia had statewide advisories for PCBs in freshwater rivers and/or lakes.

**Table 4-6. Summary of PCBs Detected in Fish Tissue<sup>a</sup> as Part of the National Study of Chemical Residues in Fish**

Congener group	% sites where detected	Maximum	Mean	Median
Monochlorobiphenyl	13.8	0.235	0.001	ND
Dichlorobiphenyl	30.7	5.072	0.021	ND
Trichlorobiphenyl	57.5	18.344	0.150	0.002
Tetrachlorobiphenyl	72.4	60.764	0.696	0.023
Pentachlorobiphenyl	86.7	29.578	0.565	0.072
Hexachlorobiphenyl	88.7	8.862	0.356	0.077
Heptachlorobiphenyl	69.1	1.850	0.097	0.017
Octachlorobiphenyl	34.8	0.593	0.017	ND
Nonachlorobiphenyl	9.7	0.413	0.003	ND
Decachlorobiphenyl	3.3	0.038	0.001	0.003
Total PCBs*	91.4	-----	1.898	0.209

\* The sum of the concentrations of compounds with 1 to 10 chlorines.

<sup>a</sup> Concentrations are in ppm ( $\mu\text{g/g}$ ) wet weight basis.

Source: U.S. EPA, 1992c, 1992d.

One state, Connecticut, had an advisory for all its coastal estuarine waters (Long Island Sound), and five states (Massachusetts, New Hampshire, New Jersey, New York, and Rhode Island) had advisories in effect for all of their coastal marine waters (U.S. EPA, 1999c). Monitoring sites in industrial and suburban/urban watersheds should be reviewed to identify sites of historical Aroclor production facilities, current and historic transformer manufacturing or refurbishing facilities, current and historic landfill and Superfund sites, and current and historic incineration or combustion facilities that are potential sources for PCB releases to the environment.

PCBs may be analyzed quantitatively as Aroclor equivalents, as homologue groups, or as individual congeners. Historically, Aroclor analysis has been performed by most laboratories. This procedure can, however, result in significant error in determining total PCB concentrations (Schwartz et al., 1987; Cogliano, 1998; U.S. EPA, 1996) and in assessing the toxicologic significance of PCBs, because it is based on the assumption that distribution of PCB congeners in environmental samples and parent Aroclors is similar.

The distribution of PCB congeners in Aroclors is, in fact, altered considerably by physical, chemical, and biological processes after release into the environment, particularly when the process of biomagnification is involved (Norstrom, 1988; Oliver and Niimi, 1988; Smith et al., 1990; U.S. EPA, 1996). Aquatic environmental studies indicate that the chlorine content of PCBs increases at higher trophic levels (Bryan et al., 1987; Kubiak et al., 1989; Oliver and Niimi, 1988).

4. TARGET ANALYTES

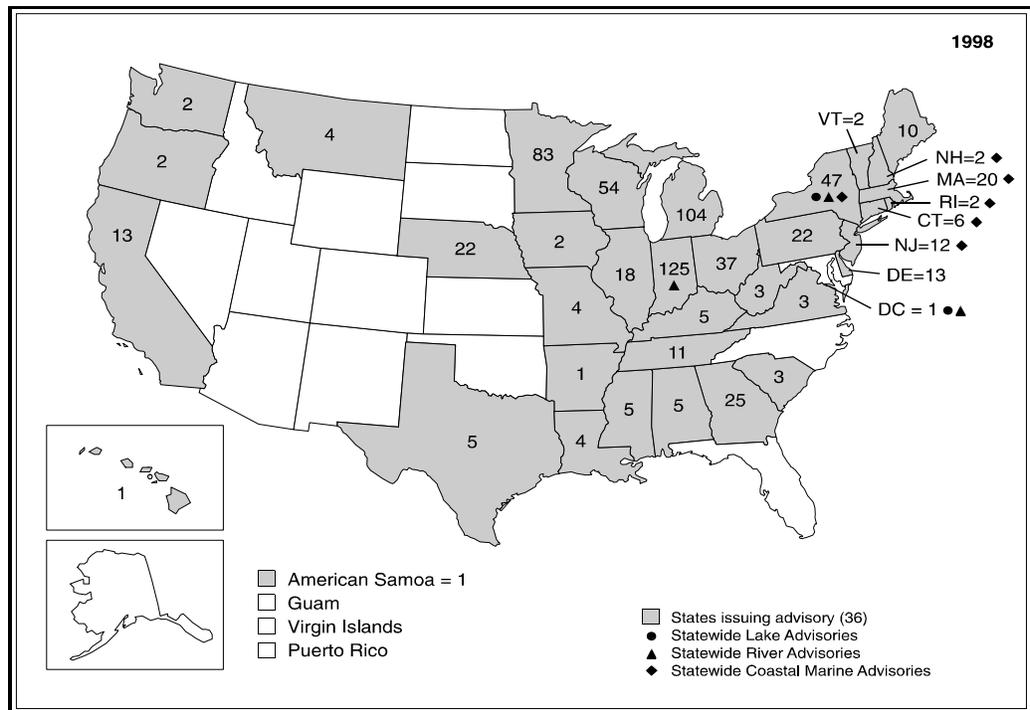
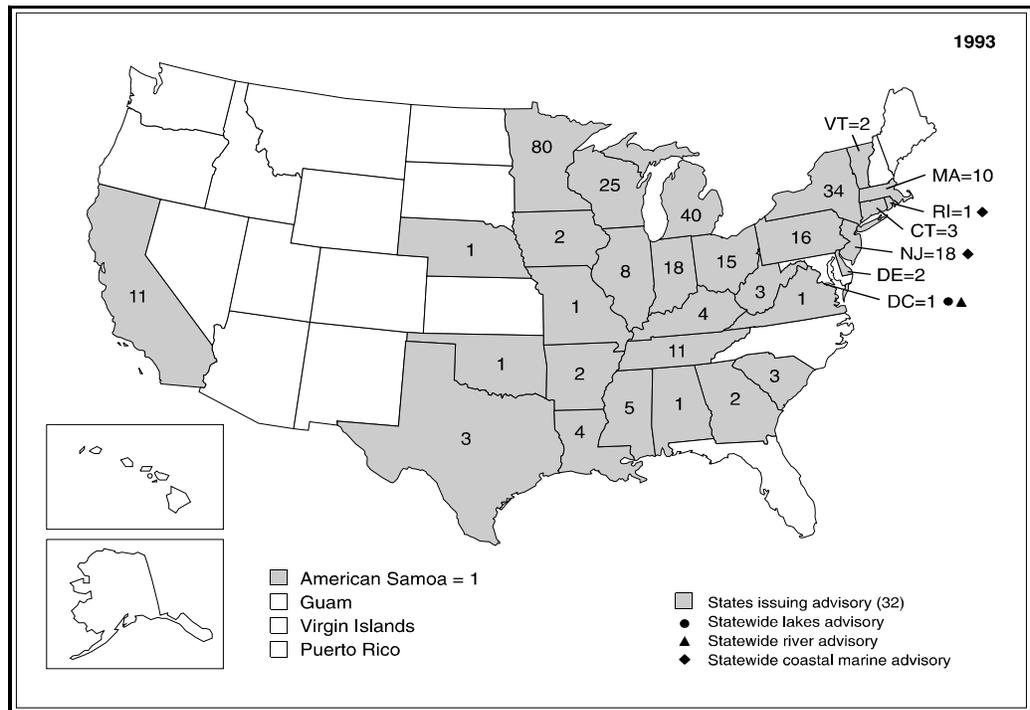


Figure 4-3. States issuing fish and shellfish advisories for PCBs.

The available data indicate that bioaccumulated PCBs are more toxic and more persistent than the original Aroclor mixtures (Cogliano, 1998). Consequently, analysis of homologue groups or congeners should provide a more accurate determination of total PCB concentrations than Aroclor analysis. PCB concentrations derived from Aroclor methods may underestimate total PCBs. In one study, the Delaware Department of National Resources and Environmental Control (DDNREC) compared results of PCBs in six fish samples as determined by Aroclor analysis (Method 608) and homologue analysis (Method 680) (Greene, 1992). On the average, the homologue method gave PCB estimates that were 230 percent higher than the results from the Aroclor method.

The major advantage to analyzing PCBs as Aroclor equivalents is that the analysis is relatively inexpensive (approximately \$200 - \$500) compared to analyzing PCBs as individual congeners (approximately \$800-\$2000). Another disadvantage to analyzing PCBs as individual congeners is that the large number of PCB congeners presents analytical difficulties. Quantitation of individual PCB congeners is relatively time-consuming. EPA has not issued a standard method for PCB congener analysis but has developed a draft method (1668) for dioxin-like congeners (U.S. EPA 1997a). This method is likely to be revised to include the capability to detect all 209 PCB congeners. Currently, only a few laboratories have the capability or expertise to perform congener analyses. Both NOAA (MacLeod et al., 1985; NOAA, 1989b) and the EPA Narragansett Research Laboratory conduct PCB congener analyses. Some states currently conduct both congener and Aroclor analysis; however, most states routinely perform only Aroclor analysis. Analytical methods for congener analysis are discussed in the following references: Cogliano, 1998; Huckins et al., 1988; Kannan et al., 1989; Lake et al., 1995; MacLeod et al., 1985; Maack and Sonzogni, 1988; Mes and Weber, 1989; NOAA, 1989b; Skerfving et al., 1994; Smith et al., 1990; Tanabe et al., 1987; U.S. EPA, 1996.

For the purposes of conducting a risk assessment to determine whether tissue residues exceed potential levels of public health concern in fish and shellfish monitoring programs, analysis of PCB congener or Aroclor equivalents is acceptable. However, because of their lower cost, Aroclor analyses may be the more cost-effective method to use if a large number of samples are analyzed for PCB contamination.

States are encouraged to develop the capability to perform PCB congener analysis. When congener analysis is conducted, at a minimum the 18 congeners recommended by NOAA (shown in Table 4-7) should be analyzed and summed to determine a total PCB concentration according to the approach used by NOAA (1989b). States may wish to consider including additional congeners based on site-specific considerations. PCB congeners of potential environmental importance identified by McFarland and Clarke (1989) and dioxin-like congeners identified by Van den Berg et al. (1998) also are listed in Table 4-7. Lake et al. (1995) and Oliver and Niimi (1988) included more than 80 congeners in their analyses of PCB patterns in water, sediment, and aquatic organisms. A recent study conducted by the DDNREC (Greene, 1999) analyzed for 75 congeners in

**Table 4-7. Polychlorinated Biphenyl (PCB) Congeners Recommended for Quantitation as Potential Target Analytes**

PCB Congener <sup>a,b</sup>	NOAA <sup>c</sup>	McFarland and Clarke (1989)		Dioxin-Like PCBs <sup>f</sup>
		Highest priority <sup>d</sup>	Second priority <sup>e</sup>	
2,4' diCB	8			
2,2',5 triCB	18		18	
2,4,4' triCB	28		37	
3,4,4' triCB				
2,2',3,5' tetraCB	44		44	
2,2',4,5' tetraCB	52	77	49	77
2,2',5,5' tetraCB	66		52	81
2,3',4,4' tetraCB	77		70	
2,3',4',5 tetraCB			74	
2,4,4',5 tetraCB			81	
3,3',4,4' tetraCB				
3,4,4',5 tetraCB				
2,2',3,4,5' pentaCB		87		
2,2',3,4',5 pentaCB	101	90	114	105
2,2',4,5,5' pentaCB	105	101	119	114
2,3,3',4,4' pentaCB	118	105	123	118
2,3,4,4',5 pentaCB	126	118		123
2,3',4,4',5 pentaCB		126		126
2,3',4,4',6 pentaCB				
2',3,4,4',5 pentaCB				
3,3',4,4',5 pentaCB				
2,2',3,3',4,4' hexaCB	128	128		
2,2',3,4,4',5' hexaCB	138	138	151	156
2,2',3,5,5',6 hexaCB	153	153	157	157
2,2',4,4',5,5' hexaCB	169	156	158	167
2,3,3',4,4',5 hexaCB		169	167	169
2,3,3',4,4',5 hexaCB			168	
2,3,3',4,4',6 hexaCB				
2,3',4,4',5,5' hexaCB				
2,3',4,4',5',6 hexaCB				
3,3',4,4',5,5' hexaCB				
2,2',3,3',4,4',5 heptaCB	170	170		
2,2',3,4,4',5,5' heptaCB	180	180	187	189
2,2',3,4,4',5',6 heptaCB	187	183	189	
2,2',3,4,4',6,6' heptaCB		184	201	
2,2',3,4',5,5',6 heptaCB		195		
2,3,3',4,4',5,5' heptaCB				
2,2',3,3',4,4',5,6 octaCB				
2,2',3,3',4,5',6,6' octaCB				
2,2',3,3',4,4',5,5',6 nonaCB		206		
2,2',3,3',4,4',5,5',6,6' decaCB		209		

<sup>a</sup> Congeners recommended for quantitation, from dichlorobiphenyl (diCB) through decachlorobiphenyl (decaCB).

<sup>b</sup> Congeners are identified in each column by their International Union of Pure and Applied Chemistry (IUPAC) number, as referenced in Ballschmitter and Zell (1980) and Mullin et al. (1984).

<sup>c</sup> EPA recommends that these 18 congeners be summed to determine total PCB concentration (NOAA, 1989b).

<sup>d</sup> PCB congeners having highest priority for potential environmental importance based on potential for toxicity, frequency of occurrence in environmental samples, and relative abundance in animal tissues.

<sup>e</sup> Congeners having second priority for potential environmental importance based on potential for toxicity, frequency of occurrence in environmental samples, and relative abundance in animal tissues.

<sup>f</sup> Van den Berg et al., 1998.

fish tissue. Of the 75 congeners, 40 were detected in every fish sample and 20 other congeners were detected in at least half the samples. The DDNREC concluded that a comprehensive target congener list is needed to account for total PCBs in environmental samples because most of the congeners contributed less than 5 percent of the total PCBs.

The EPA Office of Water recommends that PCBs be analyzed as either congeners or Aroclors, with total PCB concentrations reported as the sum of the individual congeners or the sum of the individual Aroclors. If a congener analysis is conducted, the 12 dioxin-like congeners identified in Table 4-7 may be evaluated separately as part of the dioxin risk (see Section 4.3.7). The recommendation is intended to allow states flexibility in PCB analysis and to encourage the continued development of reliable databases of PCB congener and Aroclor equivalents concentrations in fish and shellfish tissue in order to increase our understanding of the mechanisms of action and toxicities of these chemicals. The rationale for, and the uncertainties of, this recommended approach are discussed further in Section 5.3.2.6.

### 4.3.7 Dioxins and Dibenzofurans

**Note:** At this time, EPA's Office of Research and Development is reevaluating the potency of dioxins and dibenzofurans. Information provided here as well as information in Section 5.3.2.7 related to calculating TEQs and SVs for dioxins/furans has been modified since the second edition of this Volume 1 guidance was published, but is subject to change pending the results of this reevaluation.

The polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are included as target analytes primarily because of the extreme potency of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Extremely low doses of this homologue have been found to elicit a wide range of toxic responses in animals, including carcinogenicity, teratogenicity, fetotoxicity, reproductive dysfunction, and immunotoxicity (U.S. EPA, 1987d). This compound is the most potent animal carcinogen evaluated by EPA, and EPA has determined that there is sufficient evidence to conclude that 2,3,7,8-TCDD is a probable human carcinogen (B2) (HEAST, 1997). Concern over the health effects of 2,3,7,8-TCDD is increased because of its persistence in the environment and its high potential to bioaccumulate (U.S. EPA, 1987d). As of 1998, the TEF value for 1,2,3,7,8-PeCDD was changed from 0.5 to 1.0, giving 1,2,3,7,8-PeCDD and 2,3,7,8-TCDD the same toxicity equivalency factor (Van den Berg et al., 1998). 1,2,3,7,8-PeCDD is also one of the congeners that is bioaccumulated by fish (U.S. EPA, 1992c, 1992d).

Because dioxin/furan contamination is found in proximity to industrial sites (e.g., bleached kraft paper mills or facilities handling 2,4,5-trichlorophenoxyacetic acid [2,4,5-T], 2,4,5-trichlorophenol [2,4,5-TCP], and/or silvex), and municipal or industrial combustors and incinerators (U.S. EPA, 1987d), it is recommended that each state agency responsible for monitoring include these compounds as target analytes on a site-specific basis based on the presence of potential sources and

results of any environmental (water, sediment, soil, air) monitoring performed in areas adjacent to these sites. All states should maintain a current awareness of potential dioxin/furan contamination, including contamination from the 12 coplanar PCBs that exhibit dioxin-like effects.

Fifteen dioxin and dibenzofuran congeners have been included in two major fish contaminant monitoring programs; however, one congener, 2,3,7,8-TCDD, has been included in six national monitoring programs (Appendix E). Six dioxin congeners and nine dibenzofuran congeners were measured in fish tissue samples in the EPA National Study of Chemical Residues in Fish. The various dioxin congeners were detected at 32 to 89 percent of the 388 sites surveyed, while the furan congeners were detected at 1 to 89 percent of the 388 sites surveyed (U.S. EPA, 1992c, 1992d). As shown in Table 4-8, the dioxin/furan congeners detected at more than 50 percent of the sites included four CDD compounds and three CDF compounds: 1,2,3,4,6,7,8 HpCDD (89 percent), 2,3,7,8 TCDF (89 percent), 2,3,7,8 TCDD (70 percent), 1,2,3,6,7,8 HxCDD (69 percent), 2,3,4,7,8 PeCDF (64 percent), 1,2,3,4,6,7,8 HpCDF (54 percent), and 1,2,3,7,8 PeCDD (54 percent). The most frequently detected CDD/CDF compounds (1,2,3,4,6,7,8-HpCDD and 2,3,7,8-TCDF) were also detected at the highest concentrations—249 ppt and 404 ppt (wet weight), respectively. The mean concentrations of these two compounds were considerably lower, at 10.5 and 13.6 ppt, respectively. The dioxin congener (2,3,7,8-TCDD) believed to be one of the two most toxic congeners to mammals was detected at 70 percent of the sites at a maximum concentration of 204 ppt and a mean concentration of 6.8 ppt. The other toxic congener, 1,2,3,7,8-PeCDD, was detected at 54 percent of the sites at a maximum and mean concentration of 53.95 and 2.38 ppt, respectively.

The NSCRF data showed that pulp and paper mills using chlorine bleach pulp were the dominant source of 2,3,7,8-TCDD and 2,3,7,8-TCDF and that these sites had the highest median 2,3,7,8-TCDD concentrations (5.66 ppt), compared to other source categories studied, including refinery/other industrial sites (1.82 ppt), industrial/urban sites (1.40 ppt), Superfund sites (1.27 ppt), and background sites (0.5 ppt). Source categories that had the highest 2,3,7,8-TCDD concentrations in fish also had the highest TEQ values. It should be noted that OCDD and OCDF were not analyzed in fish tissues because the TEFs were zero for these compounds at the initiation of the NSCRF study. In 1989, TEFs for OCDD and OCDFs were given a TEF value of 0.001. Therefore, TEQ values presented in the NSCRF report may be underreported for samples collected at sites with sources of OCDD/OCDF contamination (e.g., wood preservers) (U.S. EPA, 1992, 1992d). It is noted that the latest TEFs for OCDD and OCDF are 0.0001 (Van den Berg et al., 1998) (see Table 5-6).

In 1993, 20 states had issued 67 fish advisories for dioxins/furans (Figure 4-4) (RTI, 1993). As of 1998, there were 59 advisories in effect in 19 states for this chemical contaminant (Figure 4-4) (U.S. EPA, 1999c). In addition, three states (Maine, New Jersey, and New York) had dioxin advisories in effect for all coastal marine waters (U.S. EPA, 1999c).

**Table 4-8. Summary of Dioxins/Furans Detected in Fish Tissue as Part of the EPA National Study of Chemical Residues in Fish<sup>a</sup>**

Congener	% Sites where detected	Maximum	Mean	Standard deviation	Median
<b>Dioxins</b>					
2,3,7,8-TCDD	70	203.6	6.89	19.41	1.38
1,2,3,7,8-PeCDD	54	53.95	2.38	4.34	0.93
1,2,3,4,7,8-HxCDD	32	37.56	1.67	2.39	1.24
1,2,3,6,7,8-HxCDD	69	100.9	4.30	9.25	1.32
1,2,3,7,8,9-HxCDD	38	24.76	1.16	1.74	0.69
1,2,3,4,6,7,8-HpCDD	89	249.1	10.52	25.30	2.83
<b>Furans</b>					
2,3,7,8-TCDF	89	403.9	13.61	40.11	2.97
1,2,3,7,8-PeCDF	47	120.3	1.71	7.69	0.45
2,3,4,7,8-PeCDF	64	56.37	3.06	6.47	0.75
1,2,3,4,7,8-HxCDF	42	45.33	2.35	4.53	1.42
1,2,3,6,7,8-HxCDF	21	30.86	1.74	2.34	1.42
1,2,3,7,8,9-HxCDF	1	0.96 <sup>b</sup>	1.22	0.41	1.38
2,3,4,6,7,8-HxCDF	32	19.3	1.24	1.51	0.98
1,2,3,4,6,7,8-HpCDF	54	58.3	1.91	4.41	0.72
1,2,3,4,7,8,9-HpCDF	4	2.57	1.24	0.33	1.30
EPA-TEQ <sup>c</sup>	NA	213	11.1	23.8	2.80

<sup>a</sup> Concentrations are given in picograms per gram (pg/g) or parts per trillion (ppt) by wet weight. The mean, median, and standard deviation were calculated using one-half the detection limit for samples that were below the detection limit. In cases where multiple samples were analyzed per site, the value used represents the highest concentration.

<sup>b</sup> Detection limits were higher than the few quantified values for 1,2,3,4,7,8,9-HpCDF and 1,2,3,7,8,9-HxCDF. Maximum values listed are measured values.

<sup>c</sup> This EPA study used TEF-89 toxicity weighting values but did not analyze concentrations of octachlorodibenzo-*p*-dioxin or octachlorodibenzofurans in fish tissues; therefore, the TEQ value does not include these two compounds or the 12 coplanar PCB congeners.

EPA = U.S. Environmental Protection Agency  
 HpCDD = Heptachlorodibenzo-*p*-dioxin  
 HpCDF = Heptachlorodibenzofuran  
 HxCDD = Hexachlorodibenzo-*p*-dioxin  
 HxCDF = Hexachlorodibenzofuran  
 NA = Not applicable  
 PeCDD = Pentachlorodibenzo-*p*-dioxin  
 PeCDF = Pentachlorodibenzofuran  
 TCDD = Tetrachlorodibenzo-*p*-dioxin  
 TCDF = Tetrachlorodibenzofuran  
 TEQ = Toxicity equivalency concentration.

Source: U.S. EPA, 1992c and 1992d.



Dioxins/furans should be considered for analysis primarily in suburban/urban and industrial watersheds at sites of pulp and paper mills using a chlorine bleaching process and at industrial sites where the following organic compounds have been or are currently produced: herbicides (containing 2,4,5-trichlorophenoxy acids and 2,4,5-trichlorophenol), silvex, hexachlorophene, pentachlorophenol, and PCBs as well as at sites of municipal and industrial waste incinerators and combustors (U.S. EPA, 1987d). EPA recommends that all of the 17 2,3,7,8-substituted tetra- through octachlorinated dioxin and dibenzofuran congeners shown in Table 4-9 as well as the 12 dioxin-like PCB congeners shown in Table 4-7 be included as target analytes.

**Table 4-9. Dibenzo-p-Dioxins and Dibenzofurans Recommended for Analysis as Target Analytes**

Dioxins	Furans
2,3,7,8-TCDD	2,3,7,8-TCDF
1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDF 2,3,4,7,8-PeCDF
1,2,3,4,7,8-HxCDD	1,2,3,4,7,8-HxCDF
1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDF
1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDF 2,3,4,6,7,8-HxCDF
1,2,3,4,6,7,8-HpCDD	1,2,3,4,6,7,8-HpCDF 1,2,3,4,7,8,9-HpCDF
OCDD	OCDF

Source: Van den Berg et al., 1998.

#### 4.4 TARGET ANALYTES UNDER EVALUATION

At present, the EPA Office of Water is evaluating one metal (lead) for possible inclusion as a recommended target analyte in state fish and shellfish contaminant monitoring programs. A toxicologic profile for this metal and the status of the evaluation are provided in this section. Other contaminants will be evaluated and may be recommended as target analytes as additional toxicologic data become available.

**Note:** Any time a state independently deems that an analyte currently under evaluation and/or other contaminants are of public health concern within its jurisdiction, the state should include these contaminants in its fish and shellfish contaminant monitoring program.

##### 4.4.1 Lead

Lead is derived primarily from the mining and processing of limestone and dolomite deposits, which are often sources of lead, zinc, and copper (May and McKinney, 1981). It is also found as a minor component of coal. Historically, lead has had a number of industrial uses, including use in paints, in solder used in plumbing and food cans, and as a gasoline additive. In the past, the primary

source of lead in the environment was the combustion of gasoline; however, use of lead in U.S. gasoline has fallen sharply in recent years due to an EPA phase-down program to minimize the amount of lead in gasoline over time. By 1988, the total lead usage in gasoline had been reduced to less than 1 percent of the amount used in the peak year of 1970 (ATSDR, 1997). At present, lead is used primarily in batteries, electric cable coverings, ammunition, electrical equipment, and sound barriers. Currently, the major points of entry of lead into the environment are from industrial processes, including metals processing, waste disposal and recycling, and chemical manufacturing and from the leachates of landfills (ATSDR, 1997; May and McKinney, 1981).

Lead has been included in five national monitoring programs (Appendix E). Lead has been shown to bioaccumulate, with the organic forms, such as tetraethyl lead, appearing to have the greatest potential for bioaccumulation in fish tissues. High concentrations of lead have been found in marine bivalves and finfish from both estuarine and marine waters (NOAA, 1987, 1989a). In 1984 and 1985, the U.S. Fish and Wildlife Service collected 315 composite samples of whole fish from 109 stations nationwide as part of the National Contaminant Biomonitoring Program (Schmitt and Brumbaugh, 1990). The authors reported that the maximum, geometric mean, and 85<sup>th</sup> percentile concentrations for lead were 4.88, 0.11, and 0.22 ppm (wet weight), respectively. Lead concentrations in freshwater fish declined significantly from a geometric mean concentration of 0.28 ppm in 1976 to 0.11 ppm in 1984. This trend has been attributed primarily to reductions in the lead content of U.S. gasoline (Schmitt and Brumbaugh, 1990). Kidwell et al. (1995) conducted an analysis of lead levels in tissues from bottom-feeding and predatory fish using the 1984-1985 data from the NCBP study. These authors reported that the mean lead tissue concentrations of  $0.18 \pm 0.37$  ppm in bottom feeders and  $0.15 \pm 0.43$  ppm in predator fish were not significantly different.

In 1993, three states (Massachusetts, Missouri, and Tennessee) and the U.S. territory of American Samoa had fish advisories for lead contamination (RTI, 1993). As of 1998, there were 10 advisories in effect in four states (Hawaii, Louisiana, Missouri, and Ohio) and the U.S. territory of American Samoa for this heavy metal (U.S. EPA, 1999c).

Lead is particularly toxic to children and fetuses. Subtle neurobehavioral effects (e.g., fine motor dysfunction, impaired concept formation, and altered behavior profile) occur in children exposed to lead at concentrations that do not result in clinical encephalopathy (ATSDR, 1997). A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. EPA's Reference Dose (RfD) Work Group discussed inorganic lead (and lead compounds) in 1985 and considered it inappropriate to develop an RfD for inorganic lead (IRIS, 1999). Lead and its inorganic compounds have been classified as probable human

carcinogens (B2) by EPA (IRIS, 1999). However, EPA has not derived a quantitative estimate of carcinogenic risk from oral exposure to lead because age, health, nutritional status, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk (IRIS, 1999).

Because of the lack of quantitative health risk assessment information for oral exposure to inorganic lead, the EPA Office of Water has not included lead as a recommended target analyte in fish and shellfish contaminant monitoring programs at this time. **Note:** Because of the observation of virtually no-threshold neurobehavioral developmental effects of lead in children, states should include lead as a target analyte in fish and shellfish contaminant programs if there is any evidence that this metal may be present at detectable levels in fish or shellfish in their jurisdictional waters.